OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

VIRGINIA MEDICAL MONTHLY

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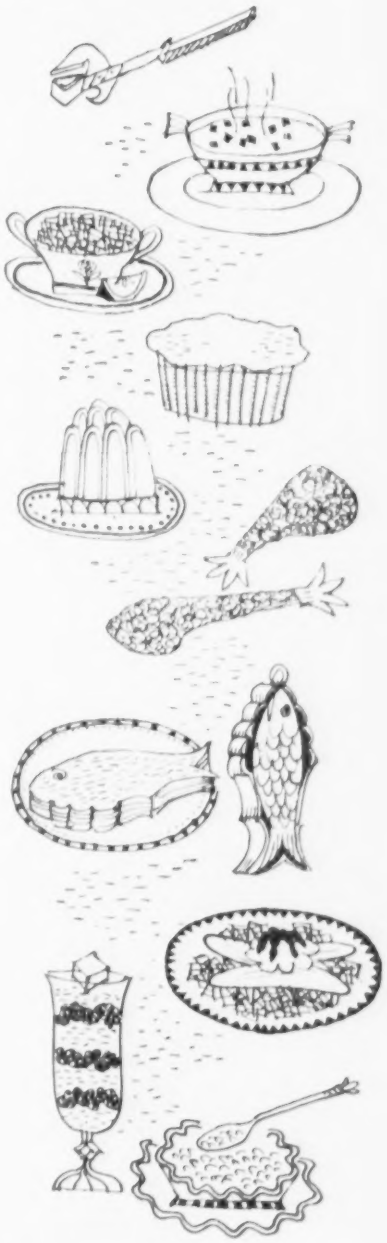

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1. Sebrell, W. H., Jr.: Trends and Needs in Nutrition, J.A.M.A. 152:42 (May 2) 1953.
2. Flour and Bread Enrichment, 1949-50. Prepared by The Committee on Cereals, Food and Nutrition Board, National Research Council, 1950.
3. Data furnished by the Laboratories of the American Institute of Baking, Chicago, Illinois.
4. Sherman, H. C.: Chemistry of Food and Nutrition, ed. 8, New York, The Macmillan Company, 1952, pp. 212, 597-600, 646.

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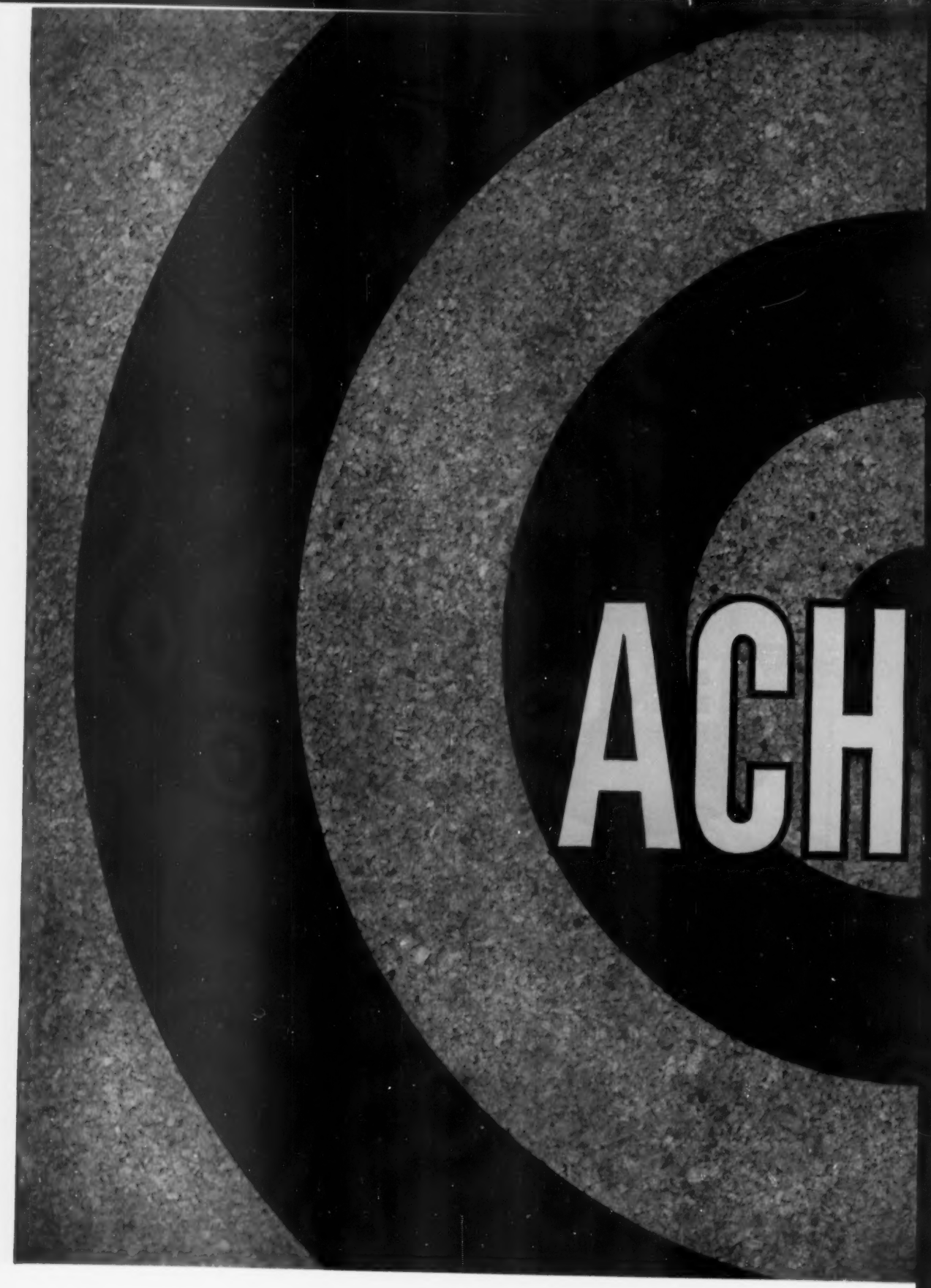
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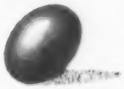

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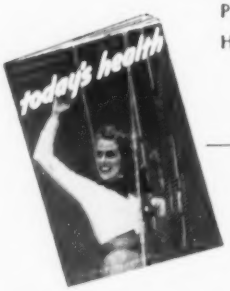

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1. American Medical Association: New and Nonofficial Remedies. J. B. Lippincott Co., Philadelphia, 1954, p. 147.

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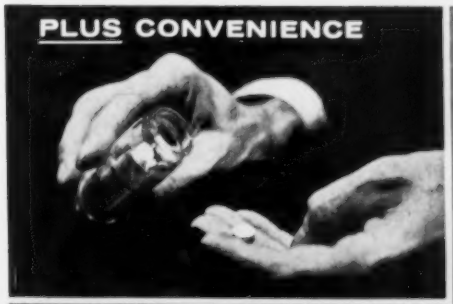

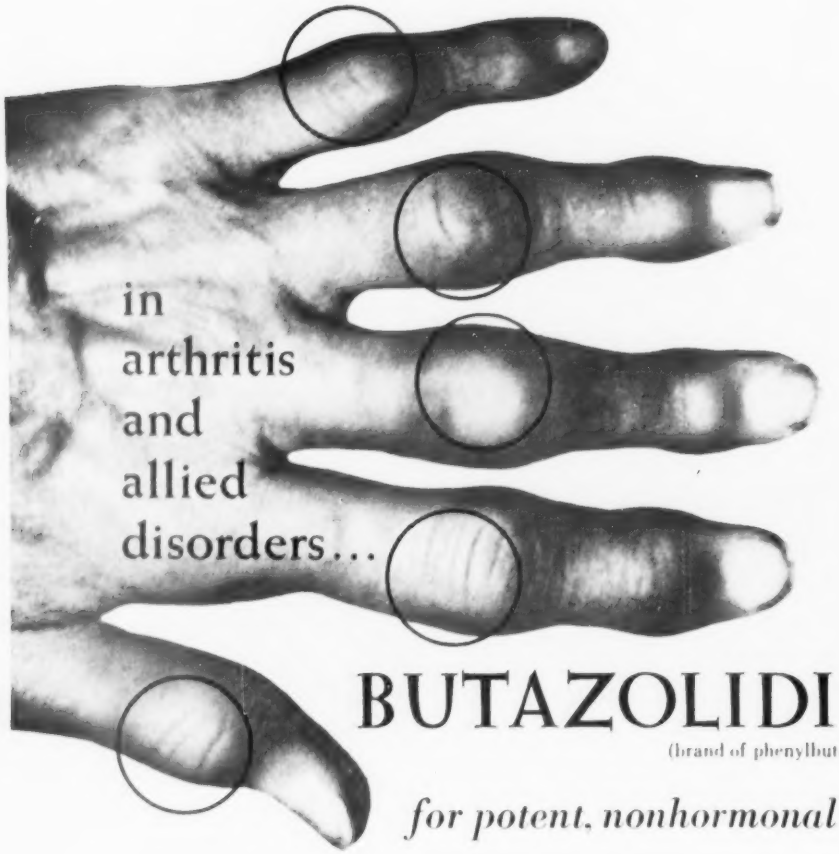

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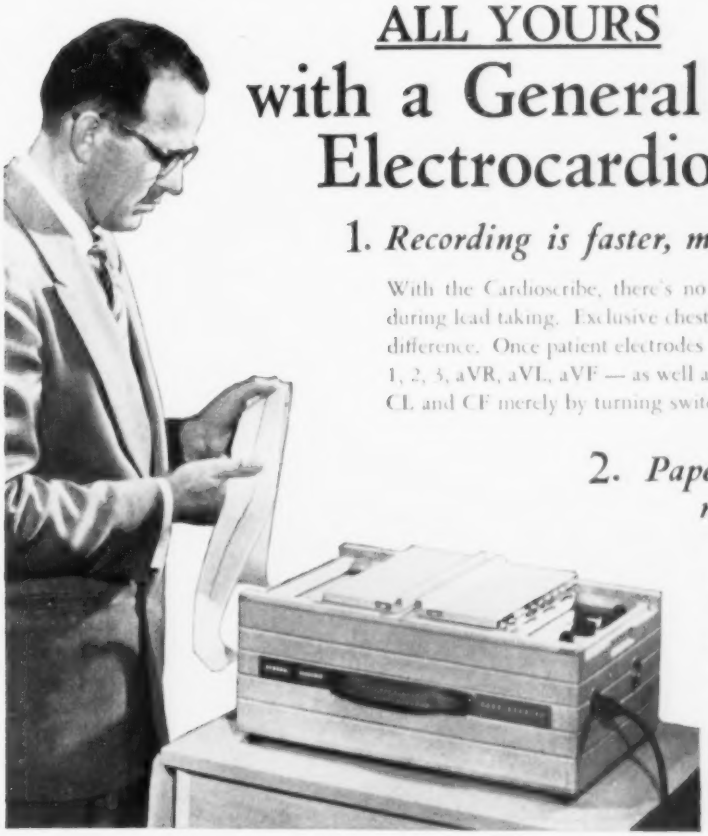

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REFERENCES: 1. Boland, E. W. and Headley, N. E., *J.A.M.A.* **148**:981, March 22, 1952. 2. Ward, L. E., Polley, H. F., Slocumb, C. H. and Hench, P. S., *J.A.M.A.* **152**:119, May 9, 1953. 3. Snow, W. B. and Coss, J. A., *N.Y. State J. Med.* **52**:319, Feb. 1, 1952.

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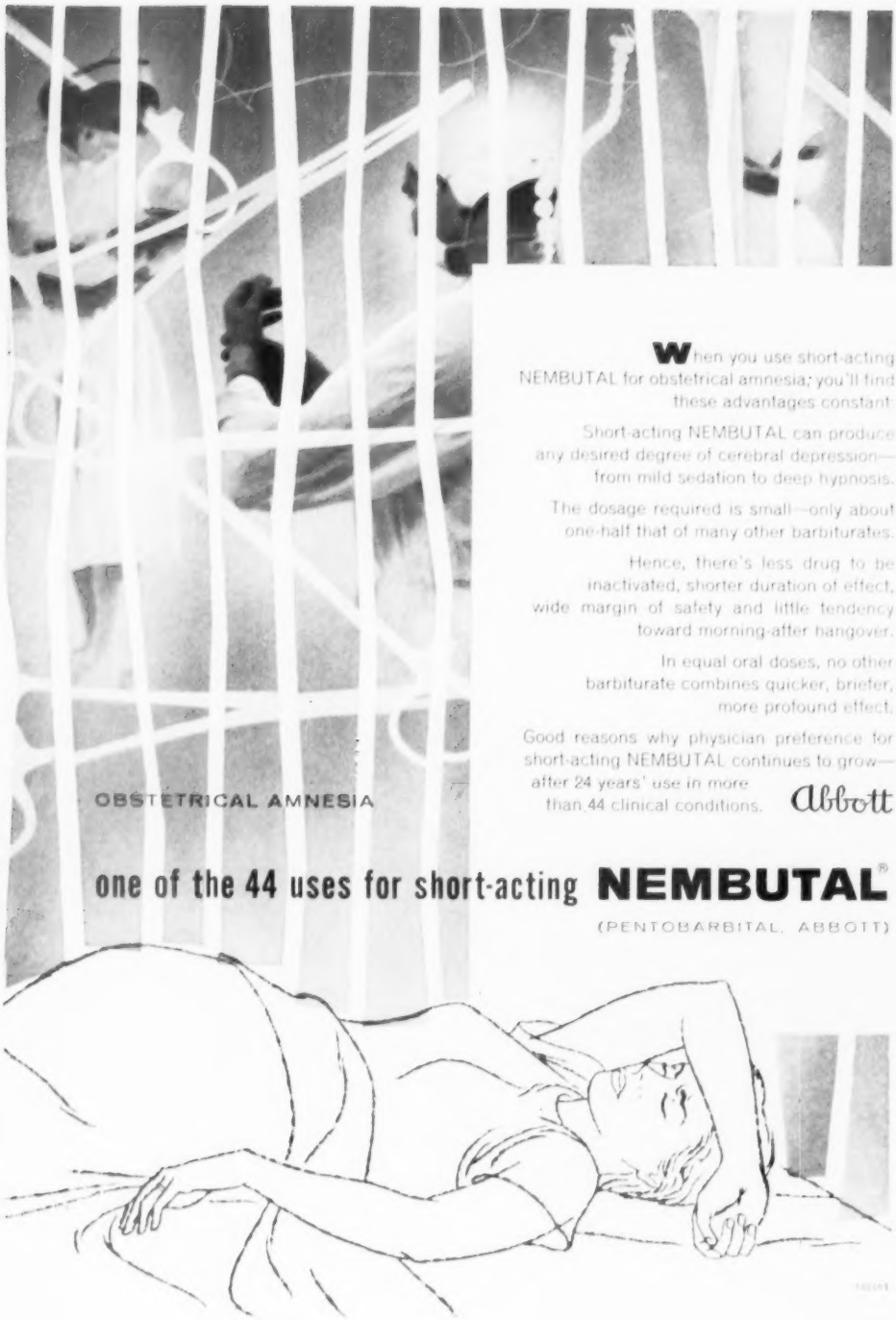
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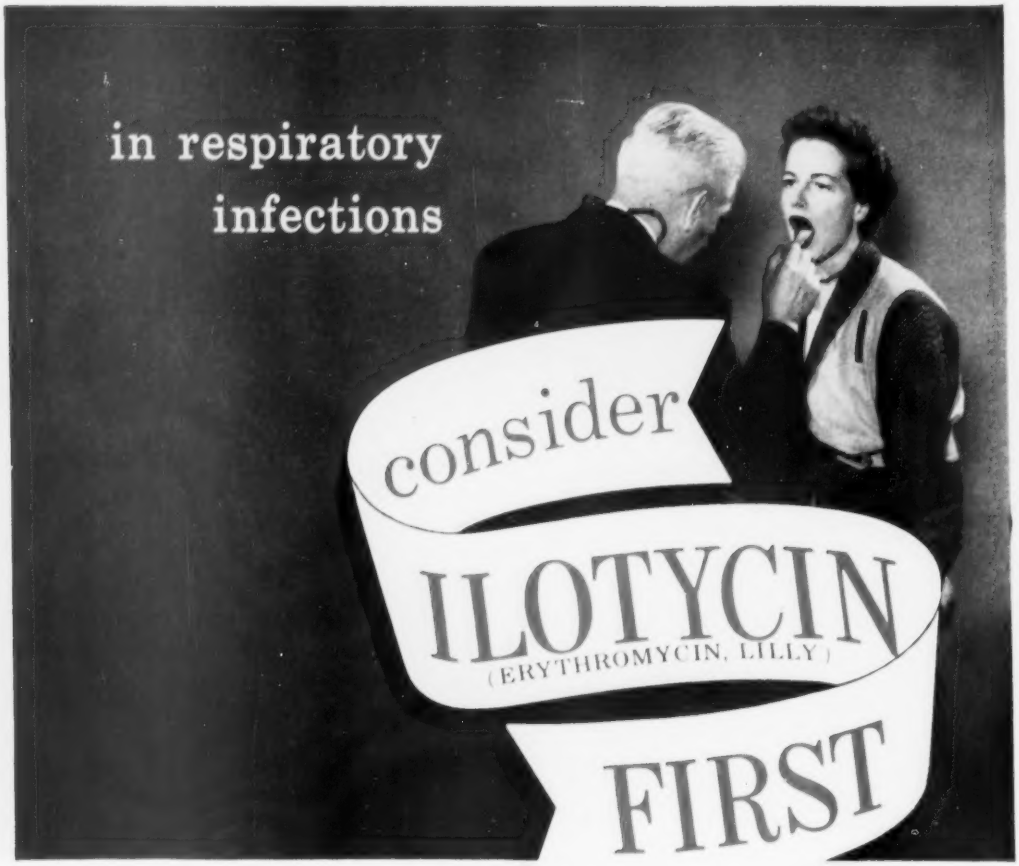

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The Virginia
MEDICAL
MONTHLY

February, 1955
Vol. 82, No. 2
Whole No. 1232

Introduction to Asthma Conference

OSCAR SWINEFORD, JR., M.D.
Charlottesville, Virginia

THIS Symposium on Asthma is the ninth of a series of Post-Graduate Conferences which have been held at the University of Virginia School of Medicine for the practicing physicians of Virginia during the past three years. This issue of the Virginia Medical Monthly is devoted to the edited recordings of that symposium. This is an innovation both for the Monthly and for the Post-Graduate Conferences. The participants are entitled to the heartiest of thanks for their effective, cheerful, and punctual co-operation, without which neither the Conference nor this record of the proceedings would have been possible.

Historical. The term asthma was coined by Hippocrates about 450 B. C. Aretaeus described the asthma paroxysm in the first century A.D. Laennec in 1819 suggested that asthma was due to bronchial spasm. Salter wrote his now famous book on asthma in 1863. Blackley demonstrated the role of pollen in seasonal hay fever and asthma in 1873. Koch reported the tuberculin reaction in 1891. Richet coined the term and studied the phenomenon of anaphylaxis at the turn of the century. Arthus described the phenomenon which bears his name in 1903. These are but a few of the milestones in the history of asthma. In the past 40 years there have been innumerable additions to the framework which these milestones provided. It is from these that the subject matter of this symposium has been accumulated.

Prevalence. Asthma and its first cousin, hay fever, were third in the 1938 Public Health Service Bulletin on the prevalence of chronic diseases, with 3,500,000 victims. Cardiovascular diseases led the list with an incidence of 7,500,000. The rheumatic diseases were a close second with nearly 7,000,000.

Asthma was diagnosed 3,779 times in this institution in a recent 5-year period. No other important diagnosis was recorded as often. Asthmatics flock to any place where "a friend of a friend of a friend" has gotten relief. This is a deplorable situation. Why should it exist? In 1952, in the Virginia Medical Monthly, I wrote "the average physician should be able to diagnose and treat the allergic diseases with the same facility and confidence with which he diagnoses and treats other diseases of the cardio-respiratory, genito-urinary, gastro-intestinal, neuro-muscular, and muco-cutaneous systems." In 1953, again in the Monthly, I wrote "this extensive experience has led to the reluctant conclusion that the average physician of today manages asthma far less effectively than it was hoped he would 20 years ago." Why is this true? Certainly it is not the fault of the physician. It is directly attributable, I think, to the failure of the curriculum committees of the medical schools to take a realistic attitude towards this problem. Certainly the allergic diseases, which affect between 5%-10% of our population, deserve more than the proverbial "lick and a promise" with which they are presented to the student and intern.

Definition. What is asthma? We define it as wheezy breathing. If people don't wheeze, they don't have asthma. In very mild asthma there may be wheezing but no dyspnea. The wheezing may be heard only by auscultation. In an attack of asthma the forces which cause wheezing are intensified, adding dyspnea to the wheezing. Attacks of asthma are

OSCAR SWINEFORD, JR., M.D., *Professor of Internal Medicine, University of Virginia, and Director of the Allergy-Arthritis Division of The Department of Internal Medicine.*

apt to be isolated in the beginning. But as time passes they tend to become more frequent and, ultimately, in many instances, some degree of wheezing becomes more or less constant. Next to wheezing and shortness of breath, cough is perhaps the third most characteristic feature. It will be discussed in some detail later. It will be emphasized that asthma is a syndrome, not a disease, and that the attacks can be caused by other things than the allergic reaction. Causes other than immunological reactions seem to play some part, usually minor, in the majority of cases. The term *bronchial* asthma is redundant and serves no purpose. All asthma is bronchial. No other organ wheezes, although some are quite noisy.

Scope of the Conference. Nothing but asthma will be discussed. The topics listed in the program have been selected arbitrarily. Discussions of prognosis, uncommon forms of asthma, and controversial topics have been omitted because time and space are limited. The program is divided into two parts. In one, emphasis is put on the mechanisms which cause wheezing. The second part is devoted to practical discussions of the principles of diagnosis and treatment of the asthmatic.

Since these proceedings are the edited transcripts of informal discussions, no effort has been made to document references to support individual statements. Each participant will be glad to supply references to any topic which the reader may wish to pursue further.

The papers are presented in the order in which they appear in the program, which is as follows.

CONFERENCE ON ALLERGY

UNIVERSITY OF VIRGINIA—

JANUARY 22, 1954

PROGRAM

INTRODUCTION—IMPORTANCE—DEFINITION

Oscar Swineford, Jr., M.D., Charlottesville.

BASIC PRINCIPLES

Pathology and Abnormal Physiology—John L. Guerrant, M.D., Charlottesville.

Immunology—Oscar Swineford, Jr., M.D., Charlottesville.

Psychogenic Asthma, Mechanisms—Richard W. Garnett, M.D., Charlottesville.

PRINCIPLES OF MANAGEMENT

Classification and Etiologic Diagnosis—Oscar Swineford, Jr., M.D., Charlottesville.

Symptomatic Treatment—Wyndham B. Blanton, M.D., Richmond.

Management of Nose, Throat, and Sinus Disease in Asthma—G. Slaughter Fitz-Hugh, M.D., Charlottesville.

Treatment of Atopic Asthma—John L. Guerrant, M.D., Charlottesville.

The Psychotherapeutic Approach—Richard W. Garnett, Jr., M.D., Charlottesville.

Treatment of Infectious Asthma—Oscar Swineford, Jr., M.D., Charlottesville.

Management of Asthma in Childhood—Susan Dees, M.D., Durham, North Carolina.

Aspiration of Secretions—George R. Minor, M.D., Charlottesville.

CLOSING PANEL

An enemy is someone you enjoy having nothing to do with; a companion is someone you enjoy doing something with; a friend is someone you enjoy doing nothing with.

Happiness is that sensation you acquire when you are too busy to be miserable.

Many a person would reach greater heights if he had greater depth.

—*Selected*

Pathology and Abnormal Physiology in Asthma

JOHN L. GUERRANT, M.D.
Charlottesville, Virginia

INTRODUCTION: When we see a patient with asthma, the first thing we notice is the fact that he is dyspneic. He is having difficulty getting air into his chest and even more difficulty getting it out. When we listen we can hear a wheeze. These gross observations suggest that there is incomplete respiratory obstruction. The obstruction is obviously not complete because the individual is getting some air in and out. The high pitched wheeze that we hear throughout the chest suggests that the obstruction is in numerous small bronchi or bronchioles instead of in the trachea and major bronchi.

It would be interesting to remove the lung from this person with asthma, give it to the pathologist, and have him describe for us the changes that have caused the difficult breathing. Since we can't do this, we will have to either produce anaphylaxis with its asthma-like manifestations in an animal and have the pathologist study the animal's lungs, or we will have to have him study the lungs from an individual who dies with asthma. Both of these studies have been done many times and from them we have a good understanding of the pathology of fatal asthma.

Information acquired in this way is very interesting, but it may not explain the difficult breathing that we see so often in patients with asthma. In the first place, anaphylaxis in animals is not the same as asthma in man, and, in the second place, there seem to be important differences between the common attacks of asthma and those from which people die. Perhaps if we want to understand why the individual with asthma is short of breath we should turn to the physiologist and ask him to study the living patient. He should be able to throw some light on the causes of respiratory tract obstruction. Certainly he can describe some of the changes resulting from the obstruction.

Pathology: Let us consider first the changes seen in the lungs of a guinea pig dying of acute anaphylaxis. They are greatly distended and when removed from the thorax do not collapse. There is increased moisture, as manifested by dripping of serosanguineous fluid from the cut surface. Microscopic study reveals emphysematous distention of the alveoli, engorgement of the capillaries, and accumulation of red cells. There is some edema in the walls of the larger bronchi. In guinea pigs that have less severe anaphylaxis and more prolonged shock, the lungs are not so enlarged and may be contracted at times. There are patches of hemorrhage on the surface. Microscopic studies shows atelectasis instead of emphysema. There is severe edema in the bronchial walls. There is a patchy infiltration of the alveolar walls, and in some lungs the alveoli contain an exudate rich in eosinophils. Even in experimental anaphylaxis the pathology is not constant.

Patients dying with asthma present a fairly characteristic appearance. On opening the chest the lungs are seen to be over distended often with localized areas of atelectasis. The distention seems to be due to actual obstruction in the bronchi or bronchioles. Even if the major bronchi are open, one still can't collapse the lung by pressure. A look at the bronchi often shows the cause of this obstruction. Whether large or small they are seen to be tightly filled with thick mucoid or muco-purulent secretions. Microscopic study often shows thickened bronchial walls. The basal membrane may be hyalinized and the wall may be infiltrated with plasma cells, lymphocytes, and eosinophils. The alveoli are emphysematous often with stretched or broken walls. Pneumonia is often present. Hyperplasia and hyperactivity of the mucous glands is often present. Hypertrophy and dilatation of the right side of the heart are seen in perhaps a third of the patients dying with asthma. Occasionally frank right-sided heart failure is present.

The major cause of death in patients dying from asthma seems to be bronchial obstruction caused by thick bronchial secretions. Common associated findings are thickening of the bronchial walls, dilated alveoli, and hypertrophy of the right side of the heart.

Do these same pathological changes occur in the common asthmatics who live on for years and years? Were these the patients that Oliver Wendell Holmes was thinking of when he described asthma as "the

JOHN L. GUERRANT, M.D., *Associate Professor of Internal Medicine, University of Virginia.*

slight ailment that promotes longevity"? I do not know, but I doubt it.

When I study the patients described in various reports on pathology in asthma and when I think of the patients I have known who died from asthma, several characteristics stand out. Most of the patients fall into one of two groups. The first and smaller group consists of patients dying suddenly and unexpectedly from anaphylaxis to a drug, serum, skin test, or allergen injection. The second and larger group is fairly characteristic and is made up of middle aged men who have had asthma for a few years or less. The asthma was almost constant, resistant to all treatment, and without evidence of significant infection or important atopy. Certainly most of our chronic asthmatics do not fall into either of these groups.

Most people believe that the structural changes in allergic asthma consist of edema of the bronchial mucous membrane, over-active mucous glands in the tracheobronchial tree, and spasm and hypertrophy of the bronchial muscle. If infection is present, the mucous membrane edema probably shows more evidence of inflammation and the bronchial secretions are purulent. If asthma has been present for very long, there is also distention of the alveoli; that is emphysema. So far as I know the blood vessels in the lung have not been well studied in asthma. Over-distention of the pulmonary vessels with associated stiffening of the lung could cause many of the symptoms of asthma.

People who die of asthma usually have bronchial obstruction due to thick bronchial secretions plus edema of the bronchial mucous membrane and thickening of the bronchial walls. We believe that people who live with asthma have edema of the bronchial mucous membrane, increased mucoid secretions in the bronchi, and spasm of the bronchial smooth muscle.

Physiological Changes: Numerous physiological observations have been made on patients with asthma. They are listed in the first table.

The lungs are over-distended. This is apparent from the physical examination as well as from the x-ray studies and laboratory studies that show increased residual volume and increased total lung capacity.

Ventilatory function is reduced. By this we mean that the patient is unable to move the normal amount of air in and out of his lung. Three observations demonstrate the reduced ventilatory function:

(1) The vital capacity is reduced.

(2) There is a reduced rate of air flow. One can measure this with an elaborate recording flow meter, with a recording spirometer that has fast-moving paper, or by simply observing how long it takes the patient to make a full inspiration and expiration.

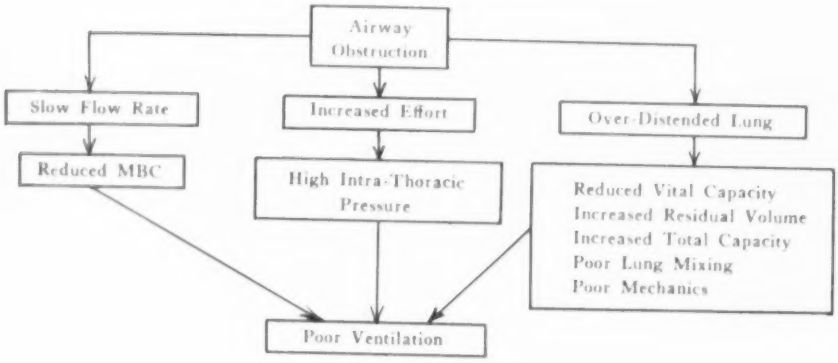
(3) The maximum breathing capacity is reduced.

TABLE 1:
PHYSIOLOGICAL CHANGES IN ASTHMA

1. Over-Distended Lung.
2. Reduced Vital Capacity.
3. Reduced Rate of Air Flow.
4. Reduced Maximum Breathing Capacity.
5. Poor Intra-Pulmonary Mixing.
6. Increased Changes in Intra-Thoracic Pressure.
7. Increased or Decreased Minute Ventilation.
8. Reduced O_2 Up-Take, Reduced Arterial O_2 Saturation, and Increased Arterial CO_2 Content when Severe.

Inspired air is poorly distributed throughout the lung. One can recognize this from the fact that it takes a longer than normal time to wash all of the nitrogen from the lung by oxygen breathing.

TABLE 2:
MECHANICS LEADING TO POOR VENTILATION IN ASTHMA



In asthma the intra-thoracic pressure swings widely. It falls quickly during inspiration and rises well above zero during expiration.

The patient with mild asthma has an increase in resting ventilation. As the asthma becomes more severe, the resting ventilation is less than normal. Along with the reduced ventilation that occurs in a severe asthma attack there is a reduction in the oxygen up-take and the arterial oxygen saturation as well as increased arterial carbon dioxide content. These changes occur only in very severe attacks.

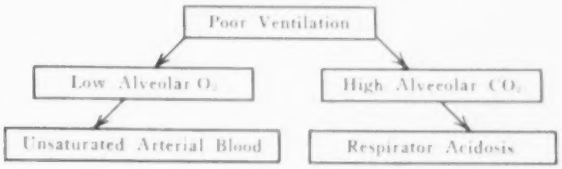
These physiological observations can be explained on the basis of one lesion; that is airway obstruction (Table 2). The obstruction causes a slow flow rate as shown by the reduced maximum breathing capacity. It leads to an increased respiratory effort and as a result the intra-thoracic pressure swings widely from inspiration to expiration. The obstruction also leads to over-distention of the lung with reduced vital capacity, increased residual volume, increased total capacity, and poor respiratory mechanics. When asthma is severe, the over-all effect of these manifestations of airway obstruction is poor alveolar ventilation.

I would like to call your attention particularly to the increased respiratory effort. That is the thing that gives the patient with asthma his symptoms. An increased effort means difficult breathing or dyspnea. So long as the patient with asthma is able to increase his effort enough to adequately ventilate his alveoli he does not get into severe trouble. When the obstruction becomes so severe that his increased effort is not sufficient to ventilate his alveoli adequately he develops hypoxia, accumulates carbon dioxide, and develops respiratory acidosis (Table 3). This occurs only when the asthma is severe.

Treatment of asthma should be directed toward relief of airway obstruction. Since serious hypoxia rarely occurs, oxygen is usually not helpful. In fact, oxygen may be harmful. There is evidence to indicate that oxygen breathing dries bronchial secretions and makes them harder to cough out. Oxygen may cause or aggravate respiratory acidosis in asthma as it does in emphysema. There is a report of one patient with asthma who developed coma on several occasions when given oxygen. Drugs that reduce respiratory effort—that is, respiratory depressants—are dangerous. Morphine is the most dangerous of all.

TABLE 3:

EFFECT OF POOR VENTILATION IN ASTHMA



Clinical Studies Listed.

The U. S. Public Health Service has published a booklet describing approximately 100 clinical studies now under way or contemplated for the fall and winter season of 1954-55 at the Clinical Center of the National Institutes of Health, Bethesda, Maryland.

The booklet gives information on procedures for referral of patients and eligibility requirements. To be admitted, a patient must be referred by a physician, and must manifest a specific condition under study by one of the seven Institutes conducting research at the Clinical Center.

Physicians may obtain a copy of the booklet by writing to the Director of the Clinical Center. Future announcements also will be sent upon request whenever new studies are contemplated.

Immunology of Asthma

OSCAR SWINEFORD, JR., M.D.
Charlottesville, Virginia

INTRODUCTION. Editing this talk has been a difficult task. The subject is a complex one. To present it as a simple topic would be misleading. To present it comprehensively would make for truly laborious reading. In an effort to solve this dilemma, the essential data have been summarized in a series of tables, while the text is made up of a sort of critical review of the subject. It is hoped that this approach will serve as a helpful guide for those who wish to study the subject more extensively, while, at the same time, serving the meat to those who do not wish to wade through the details.

Immunology has long since outgrown its early limited concern with the role of humoral factors in the prevention and control of infection. By the same token, the immunology of asthma has little to do with recovery from or protection against infection. Asthma, and other allergic diseases, are classical examples of the complete perversion of the original beneficent concept of antibodies. It is true that asthma is often mediated by antigen-antibody reactions. But instead of serving as a means of protection against the effects of harmful antigens, the allergic antibody mediates harmful reactions to otherwise harmless antigens; e.g., asthma from milk or feather pillows. Whereas this distorted immune mechanism is the commonest and best understood cause of asthma, it is important to point out that there are other causes of wheezing which will be discussed in another section.

Asthma is one of the classical allergic diseases. What do we mean by allergy? This is a long story. For our purposes, it can be considered as "a state of altered re-activity," by which we mean that the reaction of the allergic individual to the antigens of his environment differs from that of the non-allergic individual. Ordinarily the word "allergy" refers to the mechanism which produces the so-called allergic diseases, such as asthma, hay fever, urticaria, angio-edema, and serum reactions. This is the concept upon which most emphasis will be placed in this discussion. This seems simple enough at first glance. Actually, however, the problem is far from simple. Table I lists nine different types of allergic reactions which have the common background of altered re-activity plus mediation by antigen-antibody reactions. Each type of reaction, however, has its own characteristic features.

TABLE I
TYPES OF ALLERGIC REACTION

Anaphylaxis	Contact	Serum
Arthus	Atopic	Drug
Tuberculin	"Id"	Collagen

What connection does all of this have with asthma? Asthma *is not* thought to be mediated by the contact, "id", serum, or collagen type of reaction. Asthma *is* mediated typically by the atopic, conventional allergic reaction which has many features in common with anaphylaxis in the guinea pig. Asthma is also precipitated frequently by infection. The mechanism by which infection produces asthma is not well understood. In spite of this, the clinical picture of pure infectious asthma is quite characteristic. There is much to commend the idea that infection produces asthma by the tuberculin type of reaction. This idea has not been confirmed. The Arthus reaction may well play a role in infectious asthma, but too little is known about this phenomenon in man to justify further consideration here. In this clinic, more than half of a large number of chronic asthmatics had evidence of both allergy and infection.

Anaphylaxis. Most of our ideas about the immunology of asthma stem from the early studies of anaphylaxis. A sensitized guinea pig becomes dyspneic when he inhales or is injected with the antigen to which he is sensitive. The expiratory is greater than the inspiratory difficulty. If he doesn't die, he recovers completely in short order. This is the closest experimental analogue of asthma. Other similarities between the immunology of anaphylaxis and asthma as a typical example of clinical allergy are listed in Table II.

The phenomenon *anaphylaxis consists of two stages.* One is the phase of *sensitization,* which is symptomless. The other phase is the *anaphylactic reaction.*

Sensitization. An animal, the guinea pig for example, may be sensitized in two ways: (a) *Actively,* by injecting an antigen against which he makes his own antibodies; (b) *Passively,* by injecting antibodies which some other animal has made. In

TABLE II

SIMILARITIES BETWEEN ANAPHYLAXIS AND CLINICAL ALLERGY (ATOPY)

(1) Reactions highly specific
(2) Reactions mediated by antigen-antibody tissue cells
(3) Antigen apt to be inherently harmless
(4) Antibodies readily demonstrable
(5) Active sensitization lasting
(6) Passive sensitization possible
(7) Passive sensitization transient
(8) Multiple sensitizations possible
(9) Shock tissues differ
(10) Basic pathology: exudation plus smooth muscle spasm
(11) Hyposensitization possible

either case the autogenous or the foreign antibodies are thought to become attached to cells in several organs, particularly the lungs. Such an animal is said to be hypersensitive or sensitized. This animal is perfectly well until he inhales or is injected with the antigen to which he is sensitive. In this respect the actively sensitized guinea pig is quite analagous to the allergic asthmatic.

TABLE III

MECHANISM OF ANAPHYLACTIC REACTION

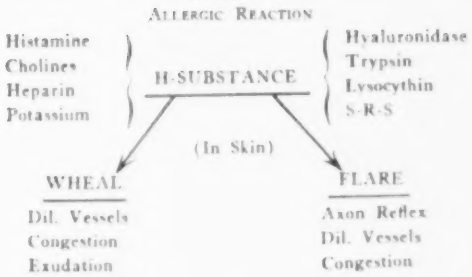
(A) Sensitization
Active
Injected antigen ——— → antibody
Tissue cells fix antibody
Passive
Injected antibody, homologous or heterologous
Tissue cells fix antibody
(B) Reaction—(when antigen is injected or inhaled)

{ Antibody + Tissue cells + Antigen } { H— Substance } ——— → Anaphylaxis

The anaphylatic reaction occurs when the guinea pig inhales or is injected with the antigen to which he is sensitive. Table III illustrates this diagramatically. The reaction occurs when the antigen-antibody-tissue cell combination occurs, the so-called allergic or anaphylactic triad. When these three things come together, profound and rapid chemical changes occur, the end-result of which is the release of several pharmacologically active materials generally referred to as the histamine-like or "H-substance". Histamine has been identified pharmacologically and has been recovered chemically from these reactions. Other substances, listed in Table IV, have also been demonstrated by students of this problem. When the H-substance is released in human skin it elicits the characteristic wheal and flare which characterize the skin-test reactions to foods and inhalants in the clinical laboratory.

TABLE IV

ALLERGIC REACTION



The Antigen may be of infinite variety. For practical purposes we classify them as foods, inhalants, drugs, or bacteria. It is customary to subclassify inhalants into pollens, epidermals, fungus spores, and domestic or occupational dusts. Anything which infects or is ingested, inhaled, or injected can cause asthma. Fortunately for the patient and the physician the majority of asthmatics are sensitive to things which are an intimate and more or less easily identified part of their daily environment. This will be discussed in more detail by Dr. Guerrant later. As a rule, antigens which cause asthma are proteins. Drugs are exceptions to this rule. The chemical nature of antigens which cause asthma has received scant and inconclusive attention. Antigens which elicit allergic reactions are called allergens.

TABLE V

ANTIBODIES CONCERNED WITH VARIOUS KINDS OF ALLERGIC REACTIONS

Reagin	Monovalant
Precipitin	Contact?
Blocking	Tuberculin?
Comp. Fixing	"Id"?

The Antibody. Table V shows that at least eight different types of antibodies are involved in the various forms of allergic reactions. Only reagin, precipitin, and the blocking antibody are germane to this discussion of the immunology of asthma, however. The antibody which mediates the tuberculin reaction is probably germane, but too little is known about it to do more than mention it here.

Strangely enough, investigation of allergic antibodies has been much more active and productive than the study of antigens. Much has been learned, but an infinite number of problems are still unsolved. A somewhat paradoxical situation will illustrate this. *The antibody which mediates the anaphylatic reaction will sensitize* guinea pigs in small doses *and protect* them in large doses. This antibody has for years been considered to be precipitin. Recent experiments, however, *seem* to have separated the sen-

sitizing from the precipitating antibody. Separation of the protecting from the precipitating antibody has not been attempted. The anaphylatic antibody with both sensitizing and protecting functions is produced in response to injections of antigen with great regularity in rabbits. The sensitizing but not the protecting function is produced easily in the guinea pig. The protecting but not the sensitizing function is produced easily in man. These paradoxes need much study.

The antibody situation in asthma seems to be quite different. There are two distinct *antibodies*, *Reagin*, which mediates the asthmatic reaction and the *blocking* or protecting antibody, which is produced in response to therapeutic antigen injections. Table VI summarizes the essential features of these two antibodies.

TABLE VI

ANTIBODIES IN ALLERGIC (ATOPIC) ASTHMA

REAGINS—SENSITIZING ANTIBODIES

Arise naturally—hard to produce
Once present, they last indefinitely
Sensitize normal tissues transiently
Allergen + regin ⇒ immediately positive skin test
Local desensitization follows reaction with allergen
Increased early in treatment
Decreased late in treatment
Destroyed by 60° C. in one hour
Highly specific
Not yet demonstrable in all cases
Bind very little allergen *in vitro*
Passive sensitization is transient, weeks
Do not permeate placenta

BLOCKING—THERMOSTABLE—PROTECTING—NEUTRALIZING ANTIBODIES

Do not occur naturally
Readily produced by injecting allergen
Do not sensitize
Do not combine with reagin
Not destroyed by 60° C. in one hour, but are in 24 hours
Combine with allergen to neutralize it
i.e., regin + tissue + { allergen + blocking antibody } ⇒ Reaction / ⇒ No reaction
i.e., compete successfully for allergen with reagin
Combination with allergen transient
Confer active and passive protection
Subside when treatment stops
Return rapidly when treatment resumed
Diffuse from injected sites within 24 hours
Do not delay nor prevent sensitization by reagin
Permeate placenta

Reagin, the Sensitizing Antibody. The essential facts about reagin are summarized in Table VI. One of the most interesting features of this sensitizing antibody is that it arises naturally. There is no known way to produce this antibody predictably. Perhaps the injection of large doses of foreign sera is an exception. When patients are sensitized by injections of antisera, precipitins usually can be demonstrated, a situation closely analogous to anaphylaxis. Perhaps penicillin will provide a more frequent exception to this rule than have the occasional examples of reagin in diabetics receiving insulin or in pernicious anemias receiving liver injections. These are examples of an artificial but unpredictable stimulus to the development of reagin.

Reagin develops insidiously from unrecognized exposures as a rule. However, there are many examples of the development of reaginic asthma from environmental exposures. The first example recognized here was the common experience of prompt relief of asthma when, years ago, kapok was substituted for feather pillows. A significant percentage of these patients developed reagins and, subsequently, asthma from the kapok exposure. Other examples are: the allergic Europeans who develop ragweed hay fever and asthma after being in this country two, three or four years; the farmers who become sensitized to hay and grain dusts; the baker who develops asthma from flour dust; and the furrier who becomes sensitive to a particular type of fur.

Passive transfer of sensitization has been the most practical use of reagin. The intradermal injection of a drop of serum which contains reagin will sensitize normal skin. If the specific antigen, ragweed extract for example, is injected into this site the next day or even ten days later, a typical wheal and flare will result. Allergists call this procedure "passive transfer". This same response will be produced immediately by the injection of a mixture of antigen and reagin-containing serum. No recognized reaction occurs from mixing the antigen and the antibody in the test tube. The reaction occurs when the allergic triad is completed by injecting the mixture into the tissues of the skin. Extensive use has been made of this passive transfer phenomenon, both in diagnosis and research.

In this clinic, passive transfer has been more useful as a research tool than in the identification of causes of asthma. There are definite limitations in the clinical usefulness of passive transfer. One is that positive passive transfer tests are rare when skin tests are negative. Another is that testing the patient's skin directly is much simpler than passive transfer tests. A third is that only a small percentage of positive reactions to direct skin tests are associated with demonstrable reagins.

Reagin persists indefinitely. Once present, there

is no known way to increase it or decrease it. Injecting antigen does not change the reagin titer significantly. Successful treatment with dust or pollen extracts, for example does not reduce the reagin titer. One observer reported disappearance of reagin after several years of treatment with enormous doses of grass pollen extract. This dogged persistance of reagin provides a prime target at which investigators have taken little or no aim to date.

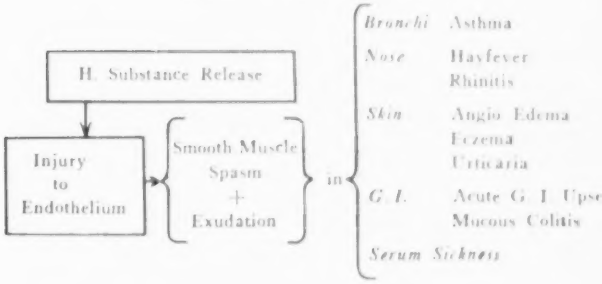
The Blocking (Thermostable, Protecting, Neutralizing) Antibody has little in common with reagin. The best known features of it are summarized in Table VI. It is called the blocking antibody because it seems to combine with the antigen in some way which prevents the antigen from performing its normal role in the allergic triad. The small diagram in Table VI illustrates this point. Nothing is known about the mechanism by which the blocking antibody competes with reagin successfully for the antigen.

The blocking antibody, produced in response to injections of antigen, confers some active protection or immunity against that antigen. This is why injections of allergen extracts are given. This antibody has been used to confer passive immunity or protection in a few clinical experiments. This aspect of treatment of reaginic asthma deserves much more study.

The blocking antibody titer subsides several weeks after antigen injections are stopped. However, the titer returns promptly, anamnestically, when antigen injections are resumed. Booster injections of allergen extracts have been used effectively in the treatment of pollen asthma. In this respect there is a close analogy to the booster injections of tetanus toxoid, typhoid vaccine and diphtheria toxoid.

Tissue Reactions. The roles of antigen and antibody in the allergic reaction have been discussed briefly. Table VII summarizes the clinical role of the tissues in this reaction. The local tissue response is basically the same. The clinical manifestation of the allergic reaction is determined by the disturbed function of the reacting tissue. Different species of animals react differently. A major shock tissue in the guinea pig is the bronchus, the pulmonary artery in the rabbit, and the hepatic vein in the dog. Man may become sensitized in most of his body systems. For example, it would not be unusual for one patient to have urticaria from aspirin, hay fever from ragweed pollen, asthma from barn dust, and nausea, vomiting, and abdominal pain from shrimp. Regardless of the site of the reaction, the basic changes in the clinical atopic type of allergy and in anaphylaxis are thought to be initiated by injury to the endothelium. The grosser changes of exudation plus smooth muscle spasm are thought to be secondary to the endothelial injury. This is the currently accepted mechanism by which the ingestion of egg, or the inhalation of ragweed pollen causes asthma.

TABLE VII
CLINICAL MANIFESTATIONS
OF
ALLERGIC TISSUE REACTIONS



Desensitization in its true sense is seldom accomplished clinically, although the word is seen commonly in the allergy literature. Perhaps a brief review of desensitization in the experimental animal followed by some clinical comments will clarify this point. Table VIII lists the four specific ways by which an allergic animal can be made to tolerate doses of an antigen which would otherwise cause severe reactions or death.

1. The repeated small subreacting doses of antigen are thought to saturate the sensitizing antibody so that it can no longer play its role in the allergic triad of antigen-antibody-tissue reaction. This can

be accomplished in hours in the guinea pig, without the production of circulating antibody.

TABLE VIII

EXPERIMENTAL DESENSITIZATION

Hypersensitive animals made refractory by

(1) Gradual saturation of fixed antibody by small dose of antigen

(2) Active production of excess circulating antibody by repeated doses of antigen

(3) Injecting excess of circulating antibody to neutralize antigen before it reacts with fixed antibody

(4) Exhaustion of fixed antibody by sublethal reactions

2. When increments of antigen, starting with small subreacting doses, are injected over a period of three or four weeks, the animal forms antibody. This antibody may be sufficient to combine with and neutralize several multiples of the dose of antigen which would have been fatal prior to the desensitizing injections. This protection is comparable to the production of the blocking antibody already discussed.

3. An allergic guinea pig can be protected from an injection of several multiples of a normally fatal dose of antigen by prior injection with an excess of antibody. This neutralizes the antigen in the same way that autogenous antibody, just discussed, will neutralize it.

4. Finally, a guinea pig which has recovered from a violent anaphylactic reaction will, for a while, tolerate many multiples of the antigen which almost killed him. Just why this should be true is not understood. The antibody is said to be "exhausted" by this reaction.

Desensitization of the tuberculin type of allergy seems to follow the first and fourth of the principles just described. *Arthus desensitization*, so far, seems to be accomplished only by injecting enough antigen to neutralize or markedly reduce the precipitin titer against that antigen.

The biggest problem in desensitization, from the standpoint of clinical application, is that resensitization recurs promptly in a few days to a few weeks after the so-called desensitizing antigen injections are discontinued. Our allergy research laboratory is attempting to find out whether desensitization with haptens (which will neutralize antibodies but will not elicit them initially nor anamnestically) will be more lasting, and therefore more desirable than desensitization with whole antigens.

An important aspect of desensitization which has been insufficiently explored is whether the increased tolerance for antigen is ever absolute. Experimental evidence to date shows only that the increase in tolerance is for a limited number of multiples of previously shocking doses of antigen.

Clinically, there are rough counterparts of each of these methods of desensitization in man. For example, one method of co-seasonal treatment of pollen hay fever is to give one to three subreacting doses of pollen extract daily. This provides marked relief in about 75% of uncomplicated pollen cases in a few days. The immunological changes which this form of treatment causes have not been studied adequately. It has been shown, in this clinic, that the titer of circulating reagin is entirely unaffected by these small subreacting doses. Tissue concentrations of reagin should be susceptible to study, but this has not been done.

The apparent capacity of the blocking antibody to prevent asthma from atmospheric pollens and dust has been discussed already.

A temporary increase in tolerance for ragweed pollen has been observed occasionally following violent systemic reactions from unintentional overdoses of ragweed extract. Perhaps this same principle is behind the good co-seasonal results which follow the administration of doses of ragweed extract which produce moderate to severe local reactions. It has been the policy in this clinic to employ small subreacting doses of pollen extract on the assumption that this carries a greater margin of safety than does the planned production of significant local reactions.

So far the discussion has been concerned chiefly with a comparison of the mechanism of anaphylaxis with the conventional wheal and flare, food and inhalant type of allergy. Certainly, this is the most commonly recognized cause of asthma.

Bacterial Allergy. Bacterial antigens produce at least four different types of allergic reactions: the anaphylactic, Arthus, tuberculin, and "id". Id reactions are the concern of the dermatologists. The Arthus phenomenon is a local reaction which requires strong antigens and precipitin titers in animals. This combination is found so seldom in man that little is known about it clinically.

The anaphylactic type of allergy to bacterial antigens, has been studied extensively in guinea pigs. The atopic, the ordinary type of allergy to bacteria in man, has received scant attention, although it does occur. A few examples are: The typical wheal and flare and passive transfer reactions to bacterial polysaccharides and nucleoproteins. The pale boggy nasal membranes and uvulae typical of the atopic type of allergy is seen occasionally in association with infected sinuses especially in children. Non-

fatal anaphylactic reactions which have followed skin tests with type-specific proteins of streptococci in rheumatic fever victims. In spite of these and quite an array of other evidence that bacteria can cause the ordinary type of allergic reaction in man and in animals, it is debatable whether infectious asthma is mediated by this mechanism. No reasonable proof that it does has been offered to date.

Tuberculin Type of Allergy. There are some striking differences between the tuberculin and the anaphylactic types of allergy. These differences, and some of the similarities are summarized in Table IX. The more relevant points need a little elaboration.

Tuberculin-type of allergy is produced, typically, by infection. It is well known that the nucleoproteins of bacteria *elicit* the tuberculin-type of reaction in animals sensitized by infection. In sharp contrast, the components of bacteria which are responsible for the *development* of the tuberculin-type of allergy (to pneumococci, for example) are not known.

Very little is known about the antibody which mediates the tuberculin type of allergy. *In vivo*, it seems to be able to sensitize a wide variety of cells in such a fashion that they will be destroyed when brought in contact with the specific bacterial antigen. This is not true of the anaphylactic nor of the atopic types of allergy.

TABLE IX
COMPARISON OF ATOPIC AND TUBERCULIN REACTIONS

Atopic	Criteria	Tuberculin
Proteins	Sensitizing Antigens	Unknown Develops in Infection (Purified Wax)
Proteins Haptens Polysaccharides	Precipitating Antigens	Nucleoproteins (Whole bacteria)
Prominent	Antibodies	Not well studied
Prompt (Min.)	Reaction time	24-48 Hours
Wheal — Transient Soft	Typical Skin Response	Tuberculin — Prolonged Nodular
High	Specificity	High
Asthma Hay Fever Urticaria, etc.	Clinical Manifestations	Local Inflamation Fever Focal Exacerbation
Eosinophiles	Cells of Exudate	Mononuclears
Possible Readily	Desensitization	Possible Readily
By Reagins	Passive Transfer	By Cells From Infected Animals
Blood Vessels Smooth Muscle	Reacting Tissue	Any Skin — Typical
Reversible — Wheal Irreversible Thrombosis — Arthus	Local Injury	Reversible if Mild Irreversible Necrotic — Thrombosis
Tissue cells + Reagin ⟹ H Subst. + Antigen	Mechanism	(a) Infection ⟹ Allergic cells (b) Allergen + Cells ⟹ Injury of cells

The familiar tuberculin skin test illustrates several characteristic features, namely: there is a delay of a day or two after the antigen is injected before the reaction becomes apparent. But once initiated, it tends to last for days. And if the antigen is too strong the patient is apt to develop a fever, an ex-

acerbation of the infection, and necrosis of the skin at the test site. The necrotic site may leave a scar when it heals. Mild reactions seem, grossly, to be reversible.

Passive transfer and desensitization of tuberculin allergy are, so far, of more experimental than clinical interest.

The tuberculin type of allergy *could* explain much that goes on in asthma due to infection. There is no direct experimental support of this statement. Lack of such evidence is an important gap in our knowledge of the immunology of asthma. The supposed role of tuberculin allergy in asthma is inferred from such evidence as the following. The tuberculin type of allergy is not limited to infection with the tubercle bacillus. Its occurence in pneumococcal and brucella infections is well known. It has been studied less extensively in staphylococcal, streptococcal, and other pyogenic infections. Attacks of infectious asthma follow exacerbations of sinusitis, bronchiectasis, pneumonia, or acute rhinitis or bronchitis. Asthma, as a rule, does not develop until 12-48 hours after the beginning of the infection. Infectious asthma is rarely transient. Once started, it is apt to last a little longer than the infection which precipitated it. Fever and other evidences of infection are typical. Chronic infectious asthma seems to lead to chronic lung disease much more quickly and severely than does ordinary allergic asthma. It is possible, even likely, that the chronic tuberculin type of reaction is responsible for this devastating complication of infectious asthma.

Summary

The immunology of asthma is complex. The mechanism of allergic asthma, the commonest form, is much better understood than is the pathogenesis of infectious and other forms of asthma.

Allergic asthma is, in many ways, analogous to the anaphylatic reaction in the guinea pig. There are important differences however.

The subject is discussed under the following headings: Different types of allergic reactions; anaphylaxis; active and passive sensitization; antigens, antibodies, and tissue reactions; passive transfer; desensitization; bacterial allergy in asthma. The tuberculin type is compared with the anaphylactic type of allergic reaction.

The subject matter has been presented in summarizing tables and, in the text, in the form of a critical review.

Fainting Is Its Own Cure.

The best way to "cure" a faint is simply to leave the victim lying flat. This is because a faint actually is a cure in itself, Dr. Alfred Soffer said. It's nature's way of giving the body a short adjustment interval to recover from sudden changes in blood movement.

"Fainting occurs when deep-lying blood vessels throughout the body, but particularly in the leg muscles and abdominal organs, are temporarily widened by paralysis or active dilation. This dilation—the result of complex nerve impulses—may be brought on by fright, shame, drug reactions, temperature sensitivity, pain and sudden blood loss.

"Contrary to popular belief, heart disease is rarely responsible for fainting," Dr. Soffer said in the November Today's Health magazine, published by the American Medical Association.

Changes in circulation may cause oxygen shortages in blood moving through the heart and brain. These make the body's protective mechanism work overtime. The body can meet this "challenge," but an adjustment period is needed. A faint and a fall to the ground is nature's way of getting stagnant blood moving back to the heart and brain. "Forcing a fainting person to sit up will only prolong the unconscious state, and may initiate irreversible damage."

He also advised loosening a victim's collar to take pressure off sensitive neck blood vessels. Giving him a drink of sweetened fluid after he recovers may help, particularly if he is sensitive to hunger faintness. But aromatic stimulants are of slight importance compared to the value of having the patient lie flat. This is nature's own wise remedy.

The Mechanisms of Psychogenic Asthma

RICHARD W. GARNETT, JR., M.D.
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THE influence of emotion upon the function of respiration is well-known from everyday experience, referred to in such expressions as, "it took my breath away" and, getting something "off one's chest" implying, to unburden oneself. The phenomena of sighing, crying, laughing and speaking are all more or less complicated expressions of feeling, involving especially the expiratory phase of respiration. In asthma, as in other vegetative disturbances which we have come to speak of as psychosomatic disorders, the pathological reaction is simply an exaggeration of the normal physiological response to a stimulus. In the case of so-called psychogenic asthma, the stimulus is a strong, and usually partly unconscious emotion which is chronic or persistent because it is not discharged or permitted a normal outlet. It is blocked or repressed because it is unacceptable or forbidden to the conscious mind.

Prior to the discovery of the allergic process, asthma was considered primarily a nervous disease and was customarily referred to as "asthma nervosa". Thomas Willis described it in detail in 1679, and stated, "the ancients allowed the cause to be only obstruction of the bronchi, but the default is partly of lungs and partly of the nerves appertaining to the breathing parts". In 1839, Allen stated "paroxysms of asthma from mental causes happen perhaps more often in physicians than in any other class of people." In 1858, Trousseau, the great French medical teacher, who himself suffered from asthma, recited in a lecture the circumstances of one of his own attacks. While in his coach barn, he detected his coachman in an act of thievery and, "under the influence of the moral emotion, my nervous system was excited and under these conditions a cause (barn dust) which under ordinary circumstances would not have caused any such effect, acted in this particular case." Fred Knight, in Boston, about 1900, summed up the three factors necessary to asthma as, (1) the neurotic habit, (2) a morbid condition somewhere in the respiratory tract whose irritation shows itself by reflex exhibition in the bronchial tubes, and, (3) the exciting factor which may be atmospheric, digestive, mental or other remote irritatives which act reflectively on the bronchial tubes.

Following the demonstration of the anaphylactic phenomenon, attention became focused on the allergic component of asthma and the older view was considered obsolete. However, as accumulated experience proved that the immunologic response did not explain all the clinical observations in asthma and as the psychosomatic viewpoint was developed, it became clear and generally accepted that it was not a question of one or the other, but both. The recognition of emotional factors in the etiology of asthma does not negate the equally well established allergic factor. Asthma is a symptom, the immediate cause of which is a spasm of the bronchioles, usually associated with edema and secretion. In the light of clinical and experimental evidence, there is no doubt that this spasm can be precipitated by exposure to a specific allergen or by more or less specific emotional situations. Either alone can produce an attack, but in most cases, both factors operate to a greater or lesser extent, varying between individuals and in the same individual at different times. Many people, with definite evidence of allergic sensitivity, do not develop clinical symptoms until the emotional factor is added to it. Some patients with asthma lose the clinical symptoms following psychotherapy or resolution of the emotional conflict while retaining the same degree of allergic sensitivity. By the same token, the clinical symptom may be controlled by treating the allergy and leaving the emotional element untouched. This is probably explained physiologically by the theory of summation of stimuli and also accounts for the therapeutic claims of both psychiatrists and allergists for their respective techniques. Going one step further, there is some evidence that the predisposition to allergic reactions in general are in some way related to personality conflict. In other words, it is possible, that sensitivity to allergens and to certain emotional trauma frequently occurs in the same person and both may be manifestations of the same constitutional factor. What, then, is the nature of these emotional stimuli, conflict situations, personality difficulties, etc., which are claimed to be of such importance in the causa-

RICHARD W. GARNETT, JR., M.D., *Associate Professor of Neurology and Psychiatry, University of Virginia.*

tion of asthma? Many clinical observers have pointed out a great variety of emotions such as sexual excitation, anxiety, jealousy and rage in fact, almost any intense emotion has been observed to precipitate asthmatic attacks, including joy. But behind this confusing array of emotions, a central physiological constellation can be discovered. The nuclear psychodynamic factor is a conflict centering in an excessive, unresolved dependence upon the mother, or mother substitute. In attempting to deal with, or defend itself against the anxiety arising from this conflict, the personality may erect many different character traits. We may, therefore, find asthmatics who are aggressive, ambitious, hostile, hypersensitive, aesthetic, timid, etc. Some are the compulsive, rigid personality organization; others are impulsive, flighty and hysterical. There is probably no very typical profile, but the repressed dependency upon the mother is a constant feature around which various character defenses may develop. This maternal dependence is different from that of the peptic ulcer cases and gastric neuroses. In the asthmatics, it is not so much the wish to be fed or provided for, but to be protected, and possessed that the person both wants and rejects and tries to repress. Every situation which threatens to separate the patient from the protective mother, or her substitute, may precipitate an attack. In children, the birth of a sibling is a conspicuous factor associated with the onset of asthma. The mothers of asthmatic children frequently are either overtly or subtly rejecting, perhaps manifested by over-protective attitudes or excessive concern for, or ambition for, their children. The child senses a lack of genuine love and security, wants it, but suppresses the wish, and experiences anxiety. He wants to cry out for his mother but he is ashamed to or afraid to, and the asthmatic attack is the somatic manifestation of the repressed impulse. In the adolescent or young adult, other factors may serve to activate the fear of loss of the mother such as the onset of sexual activity or temptation, impending marriage, leaving home to work, etc. At any age, but especially in adults, the development of forbidden hostile or aggressive attitudes toward the mother figure may threaten the dependent relationship and be the main source of difficulty. Any situation that requires the patient to manifest independent and mature behavior may revive the conflicts with his dependent tendencies and precipitate an attack.

To illustrate some of these reactions in several cases that come to mind: A young woman who now has severe asthma with recurrent periods of status asthmaticus is an only child and had her first attack shortly after graduating from high school and beginning her first job. She promptly quit her job and has lived at home with her parents since. She uses her asthma as an excuse to avoid employment or marriage.

A middle-aged asthmatic man is married but continues to live in his mother's house. The wife and mother are openly hostile and jealous toward each other and the wife puts the patient under pressure to move away but he had been unable to make the break. Attacks were frequently precipitated by emotional scenes growing out of this situation.

A middle-aged married woman had her widowed mother living with her. She resented this situation but felt obligated. Whenever she gave any evidence of irritation or resentment at the mother being in the home, the mother would assume an injured, martyred attitude which would make the patient furious but as the same time arouse feelings of guilt and frustration. Asthmatic attacks frequently occurred at such times.

A student, who had asthma from the age of 6 to 16 stated that he was quite conscious of having attacks when he felt sorry for himself and wanted some show of concern from his mother. He literally outgrew his asthma when he developed more mature reactions and discarded his childish pattern.

A 40-year old house wife was not so fortunate. She had felt rejected by her own mother in favor of a younger sister and she had reacted to this with more or less open hostility and rebellion. However, after her mother's death, she married her boss, a much older man with whom she had a very dependent relationship but towards whom she also has considerable unconscious hostility. He is a good, kind and long-suffering man, so she could not express her feelings openly but had to repress them. She routinely developed wheezing respiration at about the time he was expected to return from work in the evenings, and, whenever he went out at night to a meeting without her, she would have a severe attack and call him home to give her an injection. Thus she expressed her hostility, by her asthma, and yet avoided admitting it to her conscious awareness which would have endangered her dependent relationship. She constantly professed fear of his dying with a heart attack and yet seemed to be hastening this eventuality by subjecting him to the harrassment of her symptoms. This is a clear-cut example of unacceptable emotions (hostility) and unacceptable wishes (that her husband would die) that were

repressed but found expression through a somatic symptom.

In summarizing his material on the emotional background of asthma, French concluded that asthmatic attacks tend to be precipitated by situations that threaten to separate or estrange the patient from a mother figure. In such a situation, the asthma attacks seem to have the significance of a suppressed cry. The patient suppresses an expiratory function, such as crying, by which he would express his protection-seeking attitude towards the mother. This suppression of crying represents the wish not to give in to the dependent attachment to the mother. For some reasons, in the situations which provoked asthma attacks, the patient was unable to cry. Some of the patients stated that they had not been able to cry in years, and in some of the treated cases as the asthma attacks ceased, attacks of crying occurred instead. This was particularly apt to occur as insight into the causes was gained. The threat of loss of mother love through yielding to temptation was a very frequent factor. Many histories reveal that the mother had bound the child in a dependent relationship and at the same time rejected them at the first sign of any sexual activity or interest. Sexuality, then, became a temptation that threatened loss of the mother's love on which it was so dependent. It has been frequently observed in this connection that male asthmatic patients had continued to sleep with their mothers until the age of puberty or later. Many asthmatic patients, as children, were inclined to be overly obedient and submissive, thereby seeking to avoid rejection. Others used sickness or pain as a method for gaining and holding the sympathy and affection of the mother. Regarding the psychosomatic aspects of allergy, Karnosh rightly stated that, "no one can ignore the multifarious emotional, mental and neural phenomena which are interlocked with the allergic reaction in human tissue. Even though one cannot establish a causative relationship between allergy and nervous or mental disease, it can be said that the two afflictions are often concomitant in the same person. No allergic patient can be adequately evaluated without considering the personality structure in which the disease is implanted."

As to the physiological mechanisms involved in the actual causation of bronchial spasm from emotional stimuli, there is still much to be learned. We know that perception from both conscious and unconscious sources arising in the cortical and subcortical areas are transmitted to the hypothalamus where they apparently take on emotional significance. Thence into the autonomic pathways, the sympathetic and parasympathetic, which have some selective function. In general, asthma falls in the group of conditions associated with parasympathetic dominance. It is also spoken of as one of the conditions representing a so-called "vegetative retreat", associated with the type of personality that meets life in a passive dependent fashion with repression of aggression.

In addition to the neural, there are undoubtedly hormonal factors operating. The demonstrated symptomatic efficacy of cortisone and ACTH in asthma would indicate that the mechanisms of the endocrine system for adaptation to stress are also involved in the etiology.

TV Aids Available

A lending library of TV-tested visual aids is now available from the A.M.A. Bureau of Health Education for demonstration purposes. A booklet, "A.M.A. Televisuals," describing the anatomical charts, three-dimensional relief models and films available to medical societies without charge, may be secured from the Bureau of Health Education, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois. Put your name on the list for supplementary literature which will be issued as these "lending library" facilities expand.

Classification and Etiologic Diagnosis of Asthma

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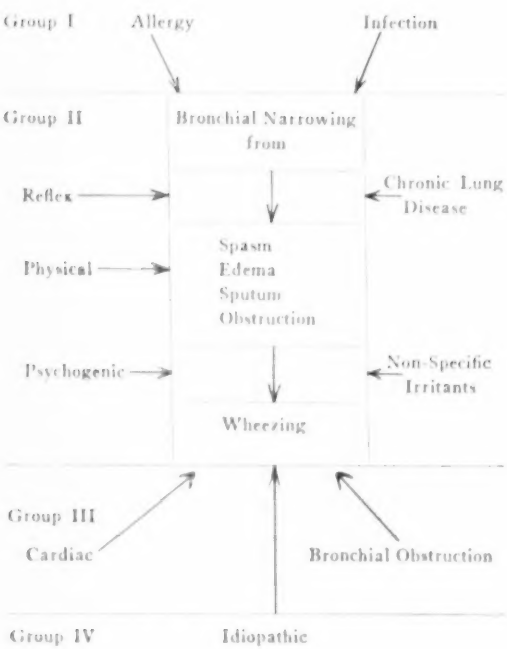
The first thing to do when a patient comes for relief of asthma is to make him comfortable. *Symptomatic treatment* will be discussed later by Dr. Blanton. The next step is to *classify the causes of his wheezing*. The information needed for this can be obtained, usually, by the history and physical examination. The third step is to attempt to *identify the individual causes of wheezing*. To do this X-ray, cardiac, pulmonary, psychiatric, clinical laboratory examinations, and skin tests may be necessary. These second and third steps will be discussed in this section. The final step is *specific treatment*, which will be discussed later.

Classification of the Causes of Wheezing: Figure I summarizes our ideas about the causes of wheezing. Notice that they are in four groups.

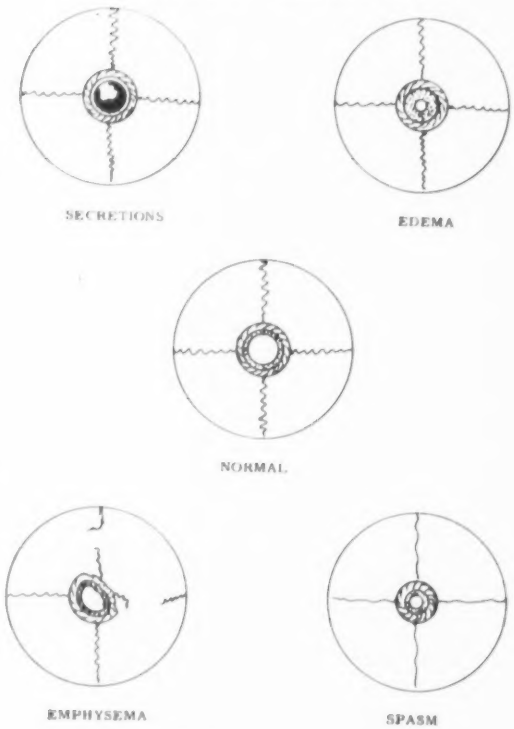
Allergy (atopy) and infection are in Group I. We think these are the two most important primary causes of wheezing. Allergy (atopy) alone caused nearly 40%, while infection alone caused about 10% of a large series of asthma cases. In more than half of this series, asthma was due to allergy plus infection plus one or more of the other causes listed in Figure 1.

FIGURE I
CAUSES OF WHEEZING



Group II: Reflex, physical, psychogenic, non-specific irritants, and chronic lung disease may serve as primary causes, but their usual role is to intensify asthma from other causes. They increase the morbidity of asthma so often that they deserve careful attention in order to provide maximum relief for the average patient.

FIGURE II
WHEEZING MECHANISMS
(Morgan E. H. - Mason Clinic Bull.)



Group III: Cardiac asthma and bronchial obstruction are usually more important in their own right than as causes of wheezing. Don't forget, however, that they may cause attacks of asthma which are hard to distinguish from those due to infection or allergy.

Idiopathic causes are in Group IV. The percent-

age in this group is small when the criteria to be discussed are thoughtfully applied.

Figure 2 simply supplements Figure 1. The normal bronchus is in the center. The lumen is narrowed by secretions (upper left), by swelling of the mucosa (upper right), by muscle spasm (lower right), and by loss of the external supporting structures of the bronchial wall (lower left).

The most common single cause of asthma is the allergic reaction, elicited by the ingestion or inhalation of things to which the patient is sensitive. We think the clinical picture of ordinary allergic asthma is so characteristic as to be easily recognized in the majority of instances. There are exceptions, of course.

What do we look for which will allow us to classify an asthmatic as allergic? Table I contrasts the clinical features of allergic with those of infectious asthma.

Allergic Asthma

The History: A *family history* of allergy merely suggests that the patient's asthma may be allergic. *Other manifestations of allergy* are the rule in allergic asthmatics. They are apt to have hives, atopic eczema, food idiosyncracies, or particularly, hay fever. The average allergic asthmatic knows that *inhaling certain things* will make him sneeze or wheeze. Everyday examples are: Pollens, dust from books, beds, rugs, basements, hay, feeds, pillows, bath powders; emanations from cats, dogs and horses. By the same token he usually *recognizes* one or more *foods* which will give him asthma. *Seasonal asthma* is usually caused by pollens in the spring, summer, or early fall, or, less typically, by house dusts in the late fall or winter.

The allergic asthmatic usually learns that he has less trouble if he avoids certain food, inhalant, and environmental exposures. But, unfortunately, he is

TABLE I

CLINICAL COMPARISON OF ALLERGIC (ATOPIC) AND INFECTIOUS ASTHMA

Allergic (Atopic)	Differential Criteria	Infectious
	History	
Predictable often	Season	Cold and changeable
Late in attack	Cough	Prominent, early
No residual	Cough	Residual
Mucoid, early or late	Sputum	Purulent
Mucoid, early or late	Nasal discharge	Purulent
Foods and inhalants	Precipitating factors	Respiratory infection
Frequent	Other allergy	Infrequent
Frequent	Sneezing	Infrequent
Frequent	Itching eyes	Infrequent
Frequent	Lacrimation	Infrequent
Absent	Fever	Common
No effect	Antibacterial therapy	Shortens or aborts
Good	Response to adrenalin and aminophylline	Fair or poor
	Physical Examination	
Pale swelling	Nasal Mucosa	Red, swollen
Mucoid	Nasal secretions	Purulent
Pale translucent	Uvula	Red, wrinkled
Pale	Tonsils	Red or normal
Pale	Lateral pharynx	Red streaks
Asthma	Chest	Asthma
Transilluminate	Sinuses	Often opaque
	Laboratory Data	
Clear or thick non symmetrical	Sinus X rays	Opaque—hazy, often unilateral
Normal	Leukocytes	Elevated or normal
Infrequent	Eosinophilia	Common
Foods and inhalants prominent	Skin tests	Foods and inhalants not prominent

apt to have trouble from other foods and inhalants which he does not recognize. It is the job of the physician to identify these. It is not necessary to identify all of the causative allergens in order to classify the asthma as allergic.

Cough has great significance in the classification of asthma. *The allergic asthmatic coughs himself* OUT OF *an attack* typically. He may cough a little during, but seldom before an attack. If he does, it isn't prominent. The end of the allergic attack is heralded by an exacerbation of the cough, which finally becomes productive of glairy, mucoid sputum.

Physical examination usually indicates that the asthma is allergic. Although we are talking about asthma, the examination of the nose and throat is often diagnostic. For example, the allergic nasal mucosa is usually pale, swollen, and perhaps boggy. Pale, grape-pulp-like polyps may be present. The throat is usually pale. Particular attention should be paid to the uvula, a greatly neglected diagnostic structure. In allergic asthma it is usually pale, swollen, and translucent at its tip. The sinuses transilluminate normally and do not discharge pus in uncomplicated allergic reactions.

Strangely enough, examination of the chest, the site of the asthma, is seldom helpful in classifying the asthma except when complications of allergic or of infectious asthma are found.

Other features which suggest allergic asthma are: The temperature is usually normal. Adrenalin and related drugs are usually quite effective. The allergic asthmatic usually feels well, for many years, when he is not wheezing. Allergic asthma is the only kind in which skin tests may be helpful.

Infectious Asthma

The clinical picture of *infectious asthma* is different. It is just as characteristic and just as easily recognized as allergic asthma. We think this concept is important. The clinical features of infectious asthma are contrasted with those of allergic asthma in Table I. Some of these differences were described in the 1920's. Others were added later. It is hard to understand why these simple but striking differences have been so little emphasized in the asthma literature.

The genesis of infectious asthma can usually be traced to an acute infection in the upper or the lower respiratory tract, from which the patient did not recover completely. The usual sequence is a post-nasal discharge, a little cough, mild stuffiness of the nose, which tend to flare-up from weather changes, fatigue, drafts, or chilling. Sooner or later, (a week, a month, a year) the cough will become troublesome and eventually wheezing will begin. Thereafter each exacerbation of the chronic respiratory infection brings on asthma. There are many variants of this typical sequence.

What do we look for which will allow us to confirm the suspicion of infectious asthma, which should be strong at this point? *Cough* is most significant. The *infectious asthmatic coughs himself* INTO *an attack.* Cough *precedes* the wheezing by minutes, hours, or days. It is *troublesome* before, during, and after the attack. The infectious asthmatic may be undecided whether the cough or the wheezy dyspnea is worse. *Sputum* may be mucoid early, but it is purulent towards the end and for some time after the attack.

Other evidences of infection, such as fever, aches, easy fatigue and the history of pus in the anterior or post-nasal discharges, may be present.

Physical Examination: Again, the nose and throat provide major leads. The nasal mucosa is not pale; it is red and swollen. The nasal space is usually bridged by mucopurulent strands, or the pus may be in the attic, floor, middle meatus, pharynx or nasopharynx. The *uvula* may be diagnostic. It is apt to be red and swollen and tends to wrinkle when the patient gags. The throat provides other clues often. For example, a velvety red swelling of the mucosa behind a posterior pillar usually means infection in the ethmoid on that side.

Transillumination may give useful information about the anterior sinuses. *This should never be omitted* from the routine examination of the asthmatic.

Examination of the chest seldom helps to differentiate allergic from infectious asthma. But a persistent patch of crepitant or moist rales on several examinations is more consistent with infectious asthma, particularly if heard between attacks.

Laboratory Examinations: Skin tests are not helpful in this type of asthma, even with vaccines, chemical fractions, or growth products of bacteria. We think *eosinophilia* is *typical of infectious and not of allergic* asthma. This is the exact opposite of the usual teaching. The sedimentation rate and leucocytes may be increased, but normal values do not rule out infectious asthma.

Infectious asthma responds to simple symptomatic therapy much less readily than does the allergic type. An attack of asthma which does not respond to adrenalin or intravenous aminophyllin, that fact alone, strongly suggests infection as the

cause. Infectious asthma is, with few exceptions, improved by antibacterial drugs, appropriately chosen and administered. This will be discussed later.

Non-Allergic, Non-Infectious Causes of Asthma

The criteria by which we recognize chronic lung disease, cardiac asthma, bronchial obstruction, and the reflex, physical, and psychogenic causes of wheezing have been discussed in detail elsewhere. Instead of repeating that discussion, it seems more appropriate to summarize in Table II those symptoms

Table II

Symptoms and Signs
Suggesting Presence of Groups II & III
Non-Allergic, Non-Infectious Causes of Wheezing

History	
Dyspnea without wheezing	Aspiration
Exhausting cough	Attacks due to
Fear of death	Heat
Interval air hunger	Cold
Known heart disease	Weather
Rapidly developing	Chest pain
Debility	Hemoptysis
Emphysema	Relief by
Seeking air for relief	Rest
Unilateral emphasis	Supine

Physical Examination	
+ A.P. diameter	Asymmetrical ventilation
Interim cyanosis	Faint heart sounds
Interim orthopnea	Inspiratory stridor
Nasal polyps	Interim reduced exercise tolerance
Sighing respiration	L. ventricle loads
Thyroid nodules	No wheezing in attacks
Venous Collaterals	

and signs which should make the physician look for other causes of asthma than allergy or infection.

A brief elaboration of the data in the tables follows:

The History. Uncomplicated allergic or infectious asthmatics are short of breath only when they wheeze. They do not sigh. They do not hyperventilate nor complain of air hunger when they are not wheezing. Nervous people do. The asthmatic who recognizes distinctly more discomfort in one lung than in the other usually wheezes from other causes than simple allergy or infection. The uncomplicated allergic or infectious asthmatic is rarely afraid he is going to die in an attack. The cardiac asthmatic is. Simple allergic or infectious asthma is not relieved by sitting or lying down. Hemoptysis is never seen in simple allergic asthma. It may occur transiently during an exacerbation of infectious asthma. It is scant then. Significant hemoptysis requires some other explanation. Rapidly developing debility or emphysema or exhausting cough are rare in allergic asthma. They may complicate infection, but when they do the infection is usually quite obvious. The cardiac asthmatic seeks air at the window or outdoors more commonly than the allergic or the infected case. Pain in the intercostal muscles after a severe attack is over is common. Chest pain during an attack is unusual in uncomplicated allergic or infectious asthma. Difficulty in swallowing as a prodrome or during an attack suggests psychogenic asthma. Subclinical allergic or infectious asthma is commonly increased to clinical severity by temperature and weather changes, drafts, etc. These are manifestations of so-called physical allergy.

Physical Examination. The physical signs of allergic and infectious asthma have been reviewed. In addition to those, the following signs should be looked for specifically in the examination of every patient who wheezes, since one or more of them are apt to be present in chronic asthma and since they imply other causes of wheezing than allergy and infection. They are: Nasal polyps, thyroid nodules, increased anterior-posterior diameter of the chest, fixation of the chest in the inspiratory position, sighing, hyper-ventilation in the absence of wheezing, cyanosis between attacks, orthopnea in the absence of wheezing, localized inspiratory stridor, dyspnea without wheezing, localized suppression or accentuation of breath sounds, asymmetrical ventilation by inspection or palpitation, faint heart sounds, the various evidences of left ventricular loads, significant reduction of exercise tolerance between attacks, signs of hyperthyroidism, cold clammy hands and feet.

There is nothing mysterious about any of this. The ability to recognize these symptoms and signs is a part of the basic equipment of every medical school graduate. If these symptoms and signs are looked for, they can be found easily. When found they mean that wheezing is due to a complication of allergy or infection or to one of the other causes.

Asthma is not a complete diagnosis. So far, attention has been focused on the clinical classification of the causes of wheezing into groups. The next step is the identification of the individual causes within the groups. This is not an unusual approach. For example, the cardiac is diagnosed, first, as having heart disease. Next, is it hypertensive, thyroid, luetic, arteriosclerotic, rheumatic, coronary or con-

genital? Further, are there arrythmias, valvular disease, enlargement, angina, infarction, or congestive failure? Similarly, meningitis is an incomplete diagnosis. Proper management requires further classification into acute and chronic, tuberculous, meningococcic, influenzal, pneumococcic, cryptococcic, viral, etc.

The diagnosis of asthma, to be complete, requires the recognition of the *types of causes* AND the *individual causes* of wheezing in each case. For example, the simplest case would be diagnosed: asthma (type of cause) allergic (individual cause) ragweed. The diagnosis of an average chronic case would be: asthma; (types of causes) allergic, infectious, reflex, physical; (individual causes) wheat, feathers, house dust, left antrum and ethmoid infection, nasal polyps, drafts, temperature changes from hot to cold. Another chronic case might be diagnosed as: asthma; (types of causes) allergic, chronic lung disease, cardiac (individual causes) milk, oak and grass pollen, bronchiectasis and emphysema; post myocardial infarction with paroxysmal left ventricular failure.

Identification of Individual Causes of Wheezing.

Recognition of the individual *non-allergic* causes of wheezing is arrived at by the use of the disciplines of internal medicine. The internist or the general practitioner should be fully as alert as the allergist to the recognition of sinusitis; psychogenic influences; nasal polyps or thyroid nodules; bronchial obstruction from carcinoma, mediastinal tumors, contracting tuberculous scars, or foreign bodies; left ventricular loads; bronchiectasis, cystic disease of the lung, silicosis, emphysema, or pulmonary fibrosis. And yet these complications have seldom been looked for in the asthmatics who come to this clinic. To explain this requires a brief digression.

Failure of the physician to recognize non-allergic causes of wheezing is due, largely, to failure of his medical school and post graduate instructors to teach him as much about asthma as they did about heart disease, diabetes, pneumonia, peptic ulcer, anemia, etc. Allergists are partly to blame also. They have over emphasized the role of allergy to a degree which encourages the concept that asthma is strictly an allergic disease. The tenor of instruction in asthma at the University of Virginia has been that, *while allergy is of major importance*, there are *other causes which must be looked for*. Failure to look for them has given rise to the cliché, which our students will remember, "When the patient starts wheezing, the doctor stops thinking." It will be a happy day for the asthmatic when medical educators realize, and accept, their responsibility for the aptness of that cliché.

The allergic reaction is, statistically, the most important cause of wheezing. The disciplines of the allergist are necessary to the identification of individual allergens. *The allergist uses the history and skin test to obtain* a TENTATIVE *list of offending allergens.*

The History. The *history form* which *follows* was designed to provide an orderly approach to the accumulation of diagnostic information from the patient. If it is filled out as indicated, most of the criteria for group classification and a number of individual causes of wheezing will be apparent in most cases.

University of Virginia Allergy Clinic

Complaint: Asthma

Other Allergy: Question by cardinal symptoms. If present mark + and discuss each briefly. Scratch out if not present. If P.I., mark P.I. and discuss under P.I.

Hay Fever		Angioneurotic edema
Urticaria	Eczema	Food idiosyncracies

Present Illness:

Onset of first symptoms: Year, Month, Acute or insidious? *precipitating factors:*

Attacks: Note—

Frequency	Severity
Duration	Prodromes
Months they occur in	Sequellae
Time of day	Fever

Freedom between attacks, partial or complete?
Effect of change of residence, occupation, etc.
Unilateral preponderance?
Cough: Precedes? Prominent?
Character nasal and bronchial discharges

List sequence of events from onset to present trouble.

Food History: Determine if any foods are related to chief complaint or to other allergy, if there are any strong dislikes, or if any particular foods cause gas, bloating, constipation, diarrhea, pain, cankers, nausea and vomiting, or mucus. Ask about all foods. Record abnormal effects from each food, i.e., milk-asthma; eggs-migraine; ? wheat-diarrhea.

Inhalant History: Note unusual effects of exposure to animals, fumes, pollen, smoke, insecticides, powders, feathers, wool; or to dusts from feeds, house, road, occupation. Record abnormal effects from each inhalant, i.e., dust-sneezing; feathers-wheezing; ? flour-cough.

Physical History: Determine effect of: seasonal changes, hot days, cold days, changes from cold to hot or hot to cold, exercise, fatigue, emotions, humidity, drafts, wet feet, dampness, sun, hot and cold baths. Record

abnormal effects, i.e., exercise-wheezing; drafts-fresh colds; ? dampness-aggravates asthma.

Focal History: Record: teeth, if dead, injured, cut with difficulty, or painful; fungus infections, sore throats, purulent nasal or bronchial discharge, excessively foul stools, constipation; or suspicious potential foci in appendix, gall bladder, tubes, prostate, or kidneys. Discuss if present.

Treatment History: List all tried. Note the effective ones.

Family History: Record allergic diseases in proper space.

Father	Mother	Brothers
Uncles	Uncles	Sisters
Aunts	Aunts	Children
Cousins	Cousins	
Grandparents	Grandparents	

Environment:

Occupation	Rug	Soap
Pillow	Upholstery	Dentifrice
Mattress	Cosmetics	House heating
Cover	Perfume	Animals
		Silica exposure

List any unusual exposures.

Systems History.

When this form is filled out carefully by a medical student studying his first case of asthma, the approach to the solution of the problems of that patient is usually quite apparent to the instructor, before any other diagnostic procedures are done. A detailed discussion of how to take an asthma history is not appropriate here, since it is available elsewhere. The importance of much of the information asked for has been brought out already.

The following typical case should illustrate the usefulness of the history.

Complaint: Asthma. A.B. white male, age 50.

Other Allergy: *Hay Fever*, 20 years, May 1 to July 1 and August 20 to October 1; *Urticaria* from peaches, peanuts and shrimp.

Onset of Wheezing: 1947, October, insidious, cause not known.

Sequence of Events: First year: wheezing, mild; followed nocturnal cough. Well in mid-summer. Wheezed with hay fever after September 1. Became severe, with cough and almost continuous stuffy nose, about October 10. Seldom comfortable more than few hours since then, except in mid-summer. Dyspnea on exertion, without wheezing, past 6 months.

Frequency: Several times daily. *Duration:* 5-45 minutes.

Time of Day: Worse at night and early A.M. Mild wheezing at any time.

Months Asthma Occurs in: All now. Worse May-June, September, late fall and winter.

Brought on by: Colds. See: food, inhalant, and physical histories.

Relieved by: Pills, midsummer, penicillin "shots" in winter.

Prodromes: Hay fever in summer, cough in cold months.

Sequellae: Tired, dyspnea on exertion, interval cough, stuffy nose.

Unilateral Preponderance: Right side nose stuffier than left.

Freedom Between Attacks: Partial in winter, complete in summer.

No Change in Environment.

Cough: Follows wheezing in spring and early fall. Precedes wheezing and is prominent and daily in winter.

Nasal Discharge: Watery in spring and in September, yellow with colds and in post-nasal drip every A.M.

Bronchial Discharge: Mucus in May and June and in early September, becoming yellow in October.

Food History: Peaches, peanuts, shrimp cause urticaria.

Inhalant History: Hay fever worse when he drives through country in hay fever seasons.

Wheezing from: dust, flufing feather pillows, moth crystals and sprays, fresh paint, and first cigarette in A.M.

Physical History: Stuffy nose from drafts. More cough and wheezing in damp rainy weather. Wheezes when laughs or runs up stairs.

Family History: Mother and one brother have hay fever.

If but do we learn from this history (which can be duplicated hundreds of times from the files of any allergy clinic)?

He is *allergic*: With hay fever, urticaria, seasonal asthma, and a positive family history for allergy.

He has *allergic asthma* from grass in May and June, from ragweed in late August and September, and from dust and feathers at other times. It is associated with hay fever. Cough follows wheezing during the pollen seasons. The sputum is mucoid then.

He has *infectious asthma*. *Cough* is *prominent* during the cold months. It *precedes* the attacks then. Asthma is precipitated by colds. The bronchial and post-nasal *discharges* are *yellow* in the cold months. The *right side* of the *nose* is stuffier than the left.

His *mixed allergic* and *infectious* asthma is aggravated by *physical* causes, such as damp rainy weather. He has wheezing from *non-specific irritants*, such as moth crystals and sprays, fresh paint, and cigarette smoke.

The role of *chronic lung disease* is strongly suggested by the morning cough, dyspnea on exertion without wheezing for six months, and wheezing from running up the stairs.

Sinus infection in the right ethmoids is suggested by the stuffy nose on the right and the purulent morning post-nasal discharge.

There is no evidence in this history of wheezing from psychogenic causes, heart failure, nor bronchial obstruction.

This case can be diagnosed, by history alone, as asthma: allergic, infectious, physical, (probably) chronic lung disease with ethmoid sinusitis. Specific

allergens are (probably) grass, ragweed, dust and feathers.

Physical examination would disclose promptly whether there were nasal polyps or evidence of sinus disease. X-ray examinations of the sinuses and chest plus simple pulmonary function studies with the vital capacity machine would verify the infectious and chronic lung disease components of his syndrome. Carefully obtained cultures of the sputum and nasal and sinus discharges would disclose the causative organisms.

The next step in the identification of individual allergens is skin testing.

Skin tests: Scratch (cutaneous) tests should always precede the intradermals (intracutaneous) because of the greater risk of anaphylaxis from intradermal testing.

Satisfactory extracts in great variety can be bought from a number of manufacturers, which Dr. Guerrant has listed.

How many and what tests should be done? This question has never been answered properly. No two allergists employ the same routines for testing. The practitioner who handles a few simple allergy problems obviously could not afford to maintain the thousand or more extracts used by the allergist. In this clinic, we test routinely with extracts of all of the ordinary foods and with about forty inhalants which are common causes of asthma. Additional tests are made when the history suggests the need for them.

A discussion of the details of skin testing is much less appropriate here than are a few pointers on the proper *interpretation* of *positive skin reactions.*

No amount of skin testing can be considered an adequate asthma investigation. *Skin tests may be of great value in providing etiologic leads* in the *allergic* patient, a large percentage of all asthmatics. On the other hand, *skin tests are of no help in the accumulation of the other data,* which are so *necessary to* the *proper management of asthma.* This should be clear, by now.

Don't assume that a reacting allergen is, necessarily, a cause of the patient's symptoms. A positive tuberculin test often does not mean *active* tuberculosis. By the same token, positive scratch or intradermal reactions to foods and inhalants do not necessarily imply, by any means, that asthma will follow exposure to them. It is probably a fair statement that our patients tolerate with complete impunity at least 80% of the foods and inhalants to which they give positive skin reactions. A food which causes a positive skin test may not cause asthma, but it may cause urticaria, indigestion, a headache, a rash, or it may cause no symptom at all. Actually, testing the skin to identify a substance which causes bronchial symptoms seems so illogical that even the limited effectiveness of skin testing is surprising.

Why go to the trouble to do skin tests when there is such a low incidence of clinical correlation between the tests and asthma? Positive *skin reactions* and the *history* provide *etiological leads.* The actual role of these individual leads has to be verified. The methods by which this is done will be discussed by Dr. Guerrant. We think the experienced allergist can help almost as many asthmatics without skin tests as with them, but it would probably take longer.

Skin reactions are frequently negative to substances (especially foods) *which can be shown, by clinical trial, to cause asthma. Conjunctival tests* with the suspected allergen are often positive, particularly in pollen asthma. More recently, the *inhalation of allergens* has attracted considerable attention as a means of identifying individual allergens. This is not recommended for general use yet, since violent attacks of asthma may be precipitated rather easily by the inhalation of allergens which are too concentrated.

The identification of foods which cause asthma but do not elicit positive skin reactions is a difficult problem. Elimination diets are an effective approach. At present we are using a "Rare Foods Diet", which consists of foods rarely eaten in Virginia. The use of elimination diets is discussed by Dr. Guerrant.

There are many "tricks of the trade" such as analyses of the enviromental dusts and fungi, short changes in residence, vacations from work and psychiatric evaluation which the allergist employs in the problem cases.

Untreated asthma tends to become chronic. Chronic syndromes require chronic treatment. Time is saved, in the long run, and results are better when the study of an asthmatic is begun with a detailed history and physical examination. This rarely takes less than an hour and a half, usually longer. Twenty-five years of intensive study of thousands of asthmatics in this clinic have failed to yield a suitable substitute for painstaking study of the problems of each cause.

Summary

The over-all problem of asthma would be simplified enormously if all cases were studied *before* their asthma became severe.

The allergic reaction to foods and inhalants is the most important single cause of wheezing. Respiratory tract infection is next. Other causes of wheezing are present in the majority of cases of chronic asthma. Examples are: Reflexes from nasal polyps; physical stimuli from heat, cold, and weather changes; chronic lung disease, such as emphysema and bronchiectasis; nonspecific irritants like smoke, paint, and moth crystals; and a variety of psychogenic influences. These tend to aggravate asthma from allergy and infection more often than to serve as primary causes.

Asthma from left ventricular failure and from bronchial obstruction may be difficult to distinguish from and may be present concurrently with asthma due to allergy or infection.

From four to six of these types of causes of wheezing can be identified in most chronic asthmatics. Criteria for recognizing these various types of causes of wheezing are outlined.

Methods are outlined by which we identify the individual foods and inhalants which cause allergic asthma in a specific case. They include the allergy history, skin and conjunctival tests, elimination diets, inhalant avoidance. These are outlined in some detail.

It is emphasized that skin tests have definite diagnostic limitations and that they are of no value in the recognition of any cause of wheezing except allergy.

Diet, Not Society.

The lower incidence of high blood pressure in certain native tribes is not necessarily due to the primitive life being simpler than ours. It might partly be because the habit of automatically sprinkling salt on food before tasting it is common only in western societies, according to two Brookhaven National Laboratory physicians. However, they noted that high salt intake is not sufficient to cause hypertension unless certain other factors also are present.

Drs. L. K. Dahl and R. A. Love, Upton, N. Y., reported they studied 1,444 persons of whom 161 never salted food at the table, 645 salted food only if it tasted unsalty, and 636 salted everything without tasting it first. Only one in the first group was found to be hypertensive, while 47 of the average group and 64 of the high-salt-intake group had high blood pressure. The intake of the low salt group was almost as low as that of the Greenland Eskimos, Australian aboriginals, mountainous Chinese tribes and the Cuna Indians of Panama. These people have been found to be virtually without any cases of hypertension. This sometimes has been attributed to their "simple" lives, but some authorities doubt that primitive life is so much simpler than ours and Drs. Love and Dahl suggested that low salt intake may have more to do with their lack of hypertension.

Their study was prompted by the discovery that among 28 consecutive hypertensive patients only one denied ever adding salt to food. With the aid of the Atomic Energy Commission, they studied every member of the laboratory staff who reported for a physical examination.

They pointed out that the study does not establish a definite cause and effect relationship but suggests that salt is one of several factors "necessary in appropriate combination" for the development of hypertension. More persons among the high-intake group were overweight than among the low or average salt-eaters. This could be because overweight persons may inherit both the tendency to heaviness and a greater susceptibility to the effects of salt, or that in eating more, they simply use more salt. It also could indicate that persons already hypertensive have a greater need for salt.

They concluded that while salt intake may be an essential factor in high blood pressure, it alone is not enough to cause the disease. The report was made in the October Archives of Internal Medicine published by the American Medical Association.

The Symptomatic Treatment of Asthma

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I.

AS BACKGROUND to the discussion of this subject, I should like to remind you that although the concept of allergy is relatively new, the word allergy is very old, and, through the ages, the remedies that have been prescribed for it are more numerous perhaps than for any other chronic disease. In the *Commentary* of Francis Adams on the seven books of the Seventh Century Paul of Aegina, the drugs and the procedures advocated by him and his predecessors are to be found. The drugs, in summary fashion, are squill, pepper, wormwood, opopanax, storax, sulphur, oxymel, myrrh, natron, opium, horehound, gum ammoniac, millepedes, assafoetida, arsenic, cardamon, hydromel, mustard, laserwort, ammoniac perfume, pine, southernwood, poley, spuma nitri, aphronitum, and castor. Other measures advocated were fomentations, fumigations, purges, blisters, cupping, bleeding, rubifacients, clysters, the cautery, burning of the head, burning balls of cotton on the chest, cold baths, friction, exercise and a long journey.

Now Paul of Aegina wrote twelve hundred years ago. Robley Dunglison, first professor of Medicine in this institution, (the University of Virginia), wrote one hundred years ago. In his *Dictionary*, you will find practically every one of those measures still surviving. Now, if you will notice the drugs that are underscored in the succeeding lists, you will find that many of them have survived even down to the present time, indicating the antiquity of some of our present therapeutic procedures.

My grandfather graduated in medicine more than a century ago. He followed the treatment of asthma advocated by his preceptor at Jefferson Medical School, George B. Wood. In his *Textbook of Medicine* published in 1847, Wood classified asthmatic therapy under emetics, antispasmodics, and narcotics; advised *ipecac*, lobelia, *stramonium*, *tartar emetic*, squill, colchicum, *ether*, musk, *morphia*, coffee, hydrocyanic acid, quinine, tobacco, and assafoetida, aided by rubifacients, cupping, leeching, holding the breath, burning nitre paper, the galvanic current, a dash of cold water, inflation of the lungs with a bellows, and a voyage.

My father graduated in medicine in 1881, one year before Henry Salter's classic description of asthma appeared in an American edition. In this work, Salter recommended *stramonium*, assafoetida, dracontium, Indian hemp, *ether*, belladonna, bromides, *sulphur*, *opium*, quinine, antimony, *ipecac*, lobelia, tobacco, *chloral hydrate*, chloroform, *tartar emetic*, squill, amyl nitrite, hot coffee, *potassium iodide*, ammonium iodide, *hydriotic acid*, potassium chlorate, *arsenic*, alcohol and hyoscyamus. He also advised breathing exercises, burning nitre paper, inhalations of compressed air and ammonia vapor, changes of residence; this recommendation runs through the whole picture. Proper posture during an attack was the particular contribution of Salter.

In 1914, while I was a medical student, Sir William Osler published his *New System of Medicine*. The drugs and procedures recommended in this volume were *morphine*, belladonna, *stramonium*, lobelia, heroin, *chloral hydrate*, amyl nitrite, tobacco, *potassium iodide*, *arsenic*, *oxygen*, and cocaine; inhalations of steam, benzoin, turpentine, ammonia, ether, and chloroform; fumes of medicated cigarettes and papers, hot hand and foot baths, the faradic current to the back of the neck, and psychiatric consultation. Noteworthy in this therapeutic armamentarium is the absence of epinephrine, ephedrine, aminophylline, although adrenalin preparations were advised for local use in the nose.

The multiplication of drugs and remedies recommended in asthma continued. Leon Unger, in his textbook on asthma, published in 1945, suggested thirty-odd non-specific therapeutic alternatives. John Sheldon, in a more recent book, gives an impressive list of the up-to-date proprietary remedies now on the market. To appreciate the number and range of them, reference must be made to his excellent text. I am glad to call your attention to the presence in it of Dr. Swineford's own quadrinal. It is a very good preparation.

A review of all the articles on the therapy of asthma in the *Annals of Allergy*, and in the *Journal of Allergy* in the last two years, not counting the reports on the use and experience with cortisone and

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ACTH, reveals more than two dozen such papers. These recent papers discuss the use of nitrogen mustard, orthoxin, pregnenolone, lobeline, the flavonoids, oral procaine, neosupirel, nephalin (pediatric), dainite, cardalin, eskel, ambodryl, triode, piromen, visammin, *sulphur* and prantol.

We must remember that the process of elimination is constantly going on, and that many drugs and procedures are still being discarded after fair and adequate trial. A few that have gone into the discard in recent years are hapamin, lipiodol, ethylene disulfonate, histamine; histaminase, ascorbic acid, ultraviolet irradiation of the blood, x-ray of the lungs and spleen, the intravenous use of alcohol.

It is difficult for one who treats asthma to escape the nostrums. Newspapers, radio, drugstore shelves, the asthmatic patient himself forcibly remind the physician of their existence and their widespread employment. Patent medicines sold for their effect on asthma depend for their action upon such ingredients as stramonium, the nitrites, iodides, and arsenic. Other ingredients are largely inert. Unger, in his book, lists seventy-six such preparations and points out the danger inherent in self-medication with expensive, often habit-forming drugs, which may in themselves become sensitizers, and by delaying the decision of the patient to consult a doctor, permit the progression of a disease otherwise reversible in its early course.

II

Present day symptomatic treatment of asthma, fortunately, is based upon a better knowledge of the pathological physiology of the disease and on the possession of a battery of pharmacologically active drugs. It is important to remember the objectives and the limitations of symptomatic treatment. This essential part of the treatment of asthma is (1) not a substitute for avoidance or specific therapy; (2) is useful while awaiting the effect of other measures; (3) is a helpful adjunct to other measures; (4) may be employed as a palliative when other measures fail; (5) finally, is indispensable in emergencies.

What are the symptoms we are called upon to combat in the treatment of asthma, especially in intractable asthma and in status asthmaticus? They may be summarized as (1) fear, nervousness, insomnia and exhaustion; (2) dyspnea from obstructed air ways, (a) by secretions (mucus or pus), (b) by a narrowed lumen due to thickened walls (edema, exudation or congestion), or to spasm of the bronchial muscles; (3) cough from secretions, edema or congestion; (4) dehydration and starvation; (5) cyanosis, (a) the manifestations of the complications of asthma.

It is helpful to recall a few theories which are basic to treatment. *First:* vagus stimulation causes spasm of the muscles of the smaller bronchi, vasodilatation and increased secretions, chiefly from the larger bronchi. Vagus stimulation may be central, truncal or peripheral. *Second:* sympathetic stimulation causes the reverse of what has been above stated. *Third:* it has to be borne in mind that there are such factors as the presence of cholinergic fibres capable of producing effects similar to those of adrenalin or epinephrine. *Fourth:* the basic concept—the antigen-antibody cellular reaction, which Dr. Swineford discussed so thoroughly this morning, with the liberation of the so-called H-substance, the predicated chief cause of bronchial spasm, mucosal edema, swelling and secretions.

What then is our therapeutic armamentarium? Briefly stated, it consists in the employment of sedatives, antispasmodics (sympathomimetics), expectorants (mucolytics), antibiotics, sulfonamides, cortisone, ACTH, breathing exercises, perhaps oxygen and helium.

Suppose we begin with the sedatives. At the head of the list stand the barbiturates with phenobarbital as a typical example. They are our stand-bys. Chloral hydrate may be employed in either an aqueous solution or in a gelatin capsule. It does not sensitize and deserves wider use. Paraldehyde, orally, or rectally, never intramuscularly, is a quick, effective and safe drug that should be tried when simpler measures fail. Seconal, nembutal, amytal and similar drugs have their place as hypnotics; occasionally, for their sedative effects. Demerol, orally or intramuscularly, is sedative, antispasmodic, and anticholinergic, but, like morphine, it is not without its dangers. Ether has proved helpful in terminating many attacks of status asthmaticus. But whether given by mouth or by rectum it should be given to full anesthesia to get effective results.

Considerable experience and judgment are required in the use of the sedatives in the treatment of asthma. The skilled therapeutist will sail cautiously between the Scylla of uncorrected emotional and nervous factors and the Charybdis of suppressed cough and an insensitive respiratory center occasioned by the over-zealous administration of depressing drugs.

Now for a glance at the antispasmodics—the sympathomimetics. Epinephrine is at the head of this list. As you are aware, it may be given subcu-

taneously, intramuscularly, intravenously or intrabronchially. It is still in my estimation the most valuable drug in the treatment of asthma. Although it can be given in a number of ways, unfortunately it cannot be given by mouth. In modified form we have susphrine, effective in small doses with prolonged effect and devoid of some of the side reactions of epinephrine. For a number of years ephedrine has been generally employed in the treatment of asthma administered orally or by hypodermic injection. Its chief advantage is its oral effectiveness, but it is slow and produces nervousness which has to be counterbalanced by combining with it various sedatives. Isuprel is employed in linguets or by aerosol, but it cannot be counted upon except in the milder cases of asthma. Aminophylline lends itself to a variety of methods of administration, being effective orally, intravenously, by rectal instillation, or in rectal suppositories. Until recently its administration by mouth has been disappointing, but, fortunately, we now have cardalin and damite combinations containing aluminum hydroxide and ethyl aminobenzoate enabling us to give without side effects doses as large as five grams.

Now for the expectorants and the nauseants. Out of a long list we must limit ourselves to a few effective drugs. Potassium iodide should head the list, employed in doses of from 5-20 grs., three or four times a day, after meals, in a full glass of water or a glass of milk. Sodium iodide may be equally good, used, of course, intravenously; but there is never in my estimation, or rarely, any use for the intravenous medication with the iodides since iodine by mouth is so readily absorbed. Ammonium chloride in uncoated 5 gr. tablets ranks next. The uncoated tablet, because of its nauseant effect on the stomach, is preferred. Hydriotic acid in syrup form is popular in pediatric practice in dram doses three or four times a day. Our pharmacologist tells us that it is a respiratory depressant and should not be used. Expectorants which are classified as nauseants include ipecac, the syrup of which is given in 0.5 gm. doses, tartar emetic (Syrup of Squill) gr. 1/20-1/10, and apomorphine, gr. 1/30 by hypodermic injection.

Useful expectorant mixtures that have recently come on the market are benadryl expectorant, pyribenzamine expectorant, and phenergan expectorant. We have little use for the popular preparations of a generation ago. Stokes' mixture, brown mixture, or Coxe's hive syrup.

We come next to a discussion of the inhalants—the so-called mucolytic agents. Steam is, of course, an old-fashioned method of obtaining the desired end. It may be inhaled with or without benzoin or other substances. Some allergists I know oppose the use of benzoin altogether. Alevaire, an aqueous solution of triton, is a detergent effective in lowering viscosity, as it thins mucopurulent exudates. It is administered by an aerosol nebulizer capable of delivering a fine mist. It is supplied in bottles containing 500 cc. which is usually enough to last twenty-four hours. It may be employed alone or as a vehicle for other agents but never with terramycin. Tryptar, the Armour Laboratories preparation, is a pure crystalline form of trypsin, and may be administered trans-orally by tube or by mask, 15 minutes at a time, once or twice a day. It is supplied in packages containing 125,000 units of trypsin per vial with an ampule containing 5 cc. of diluent.

We have now to consider the antihistaminics, of which there are a great many—thirty-odd to be exact. They are administered orally, in doses of from 4-100 mg. three or four times daily, or by aerosol, theoretically the most effective method, since it brings the drug directly into contact with the shock organ, or by injection—there are six which are injectable (thephorin, 12.5 mg.; decaprynminergic 5 mg.; dexapyramine (vitamix) 25 mg.; pyribenzamine 25 mg.; histadyl 20 mg.; benadryl 10 mg.). It is to be remembered that the intravenous effect of these drugs is supposed to be largely one of sedation. We do not employ them in this manner. If one is going to use these preparations, one should remember that chemically and pharmacologically they fall roughly into four classes, with benadryl, pyri-benzamine, chlor-trimeton, and thephorin as examples of each class, and in practice one should try each in succession before deciding which class is most effective and most devoid of side reactions. On the whole, it should be said that the antihistaminics are not for asthmatics. In our experience they are disappointing, being only from 0-50 per cent effective at best. They may actually prove harmful by drying the mucous membranes.

We have at our command two additional powerful agents, remarkably effective in the treatment of asthma—cortisone and ACTH. Cortisone is available for oral use in 5 and 25 mg. tablets; for injection in aqueous suspension in two strengths—1 cc. containing 25 mg., and 1 cc. containing 50 mg. It may be employed in one of three ways—in an emergency as in status asthmaticus when descending

daily doses of 300, 200, 100 and 50 mg. may be given; or intermittently in recurring asthmatic attacks, varying the dose according to the severity of the attack; or continuously in intractable asthma. In this manner it has been given for one or two years daily, in 50-100 mg. doses, with good results. In a Boston experiment 90 per cent of the patients so treated remained in a satisfactory state of health with few side effects. The chief difficulty in the prolonged use of cortisone lies in the identification and management of concurrent infections.

ACTH is equally effective in the treatment of asthma. It may be given intramuscularly in 25-50 mg. doses every 6 hours, or as HP ACTHAR Gel which is now getting to be a popular method. This new preparation is easily administered in 20-40 mg. units per cc. once a day. With improvement the necessity for injections becomes less frequent so that one or two a week suffice for maintenance therapy. ACTH may also be given intravenously by continuous slow drip in doses of 10-20 mg. every 24 hours. Both of these agents, cortisone and ACTH act dramatically. We prefer cortisone because it can be given by mouth.

Cough has been referred to as a troublesome symptom in asthma. The allergic cough is due, as other coughs are, to stimulation of the afferent nerve endings by secretions and or mucous membrane swelling. We presume the most sensitive area in the tracheo-bronchial tree is the bifurcation of the trachea, but of course cough can originate in the pharynx, the larynx, the trachea, the bronchi, the lung parenchyma, or the pleura. There are three kinds of cough that we are interested in now: the cough of allergic tracheitis, which is usually pre-asthmatic, usually occurs in children, and usually occurs at night; codeine is not advocated, but ephedrine, or one of the antihistaminics, is. There is the bronchial cough in which the patient tries to cough himself *out* of an attack of asthma. Here codeine should not be given, but epinephrine, ephedrine, the iodides, or the antihistaminics. Finally, there is the bronchial cough in which the patient coughs himself *into* an attack of asthma, and here codeine is the drug of choice.

It is necessary in the successful treatment of asthma to take into account some further considerations. First, the type of disease—whether or not it is the atopic type of asthma or whether it is the infectious type of asthma. Treatment has to be directed according to the etiology. We have also to consider the severity of the disease and to be guided by whether we are treating the patient during an attack or between attacks, and if during an attack, whether it is a mild, a moderate, or a severe attack such as status asthmaticus. Finally, we have to give consideration to the complications of asthma.

And now a last word of caution. Morphine and its derivatives—never! And this includes demerol. Aspirin, only to non-sensitive patients. I should say that in the employment of drug therapy in asthma one should always be careful to discover in advance whether or not the patient is sensitive to that particular drug by giving it in small quantities. Oxygen has disadvantages that outweigh its advantages, and should be reserved for cyanosis only.

The results of symptomatic treatment I should say are good: they are very good sometimes, but they parallel the diagnostic ability and the therapeutic skill of the doctor and, not least in importance, the cooperation of the patient.

Don't Let Those Tax Forms Get You Down!

If higher mathematical equations . . . dates like April 15 . . . or mountains of tax forms . . . get in your hair, doctor, watch for the series of helpful articles on taxes appearing in the *Journal* of the AMA during January and February. Prepared by the Law Department staff, these articles especially pinpoint the changes in the internal revenue code as adopted by the 83rd Congress. These articles later will be incorporated in a handbook on taxes which will be available from the Law Department.

Treatment of Sinusitis in Patients with Asthma

G. S. FITZ-HUGH, M.D.
Charlottesville, Virginia

TIME does not permit a discussion of the symptoms and signs of sinusitis as related to asthma, the bacteriology and immunology of the sinuses, and the histopathology of the nose and paranasal sinuses in allergy. It should be mentioned, however, that the pathologic changes in the membranes of the nose and sinuses will be mainly edematous and cystic as the result of allergy, with, possibly, some additional degenerative manifestations in certain cases. Superimposed or initial infection may, of course, alter the picture.

My discussion will be limited to brief remarks in regard to the treatment of rhinitis and sinusitis associated with asthma. One basic concept must be recognized, which is, that asthma is not the cause of sinusitis, and, in turn, sinusitis is not the cause of asthma. In many instances, the two problems must be solved together, realizing that often the sinusitis and asthma are caused by the same factors.

There are differences of opinion concerning the incidence of sinusitis in asthmatics and in those patients with an allergic rhinitis. When we examine the literature, we find some who state that the incidence is low, and others who believe it is high. Reasonable and accurate figures show the occurrence of gross changes in the mucous membranes of the nose and sinuses, in patients with nasal allergy without asthma proper, to be high, say, 85 to 90 per cent. Again, a fair and reasonable figure for the incidence of gross membranous changes in the sinuses of all asthmatics, infectious and non-infectious, would be near 75 to 80 per cent. In other words, there will be some sinusitis associated with asthma in 75 to 80 per cent of the cases.

TREATMENT OF SINUSITIS
IN PATIENTS WITH ASTHMA

SLIDE 1
NON SURGICAL TREATMENT OF RHINITIS,
SINUSITIS, & PHARYNGITIS

Desensitization
Antihistamine Therapy
Vasoconstriction
Hormonal Therapy
Antibiotics & Sulfonamides
Irradiation
Miscellaneous Drugs

G. S. FITZ-HUGH, M.D., *Professor of Otolaryngology, University of Virginia.*

After these preliminary remarks, I should like to begin that part of this discussion to which I have been assigned; the treatment of rhinitis and sinusitis associated with asthma. Please refer to the first slide or chart.

Desensitization: This form of therapy, as far as we, the otolaryngologists here, are concerned, is the responsibility of the allergist. We refer our patients requiring this treatment to the allergists, and take no active part here, except occasionally to administer the recommended injections.

Antihistamines: We have found antihistamines to be very beneficial, and these drugs are freely prescribed, particularly in the nasal allergies. They certainly, in many instances, give comfort to the patient. We also use antihistamines in cases with nasal complaints, but with no obvious allergy. They are given as a trial therapeutic test, and one is sometimes surprised with the good results obtained. Actually, I prescribe antihistamines in the treatment regime of plain common coryzas, on many occasions with good effects. Scientifically, antihistamines have been shown to be of no value in influencing the course of a viral infection; but, undoubtedly, in many of these infections, an element of allergy exists, and the antihistamines are of aid in controlling this. Also, as far as I have been able to determine, no harm results from the use of an antihistamine in coryzas.

Vasoconstrictors: A word of warning should be noted here. Nasal drops containing drugs causing vasoconstriction of the vessels of the nasal mucous membranes are used too frequently. The rhinologist, considering the physiology of the nasal mucous membranes, prefers an aqueous vehicle containing a moderate vasoconstrictor, such as a solution of neo-synephrine 1/4%, or ephedrine sulphate 1%. There are many other nasal constrictor solutions on the market that have little or no advantage over those mentioned. Many drugs are incorporated in the various nasal solutions, such as antibacterials, hormones, and antihistamines; their value is questionable. Anyone considering nasal physiology and pathology will be aware of the limitations in the effectiveness of topical medications. Again, one must consider the possibility of becoming addicted to nasal medications. In addition, certain patients

develop a sensitivity to nose drops, resulting in a so-called "kick back" reaction to the drops, which is severer than the initial reaction. Often by administration of an antihistamine and barbiturate, especially at night, plus abstinence from the use of nasal drops, one may accomplish good results in a few days, in troublesome cases of vasomotor and allergic nasal congestion. Any nasal medication should be prescribed for a stated and limited period.

Hormone Therapy: The use of cortisone and ACTH, administered orally, is particularly helpful in interrupting an acute attack of allergic rhinitis. We have utilized cortisone very successfully in this manner. We do not use, or advocate the use of, cortisone for injection into the nasal structures or nasal polyps. Investigations have shown that this method of therapy is of no value, and actually is dangerous. One hundred mg. of cortisone may be given three times a day, gradually decreasing the doses over a period of five to seven days, to tide a patient over an acute period of nasal discomfort.

Antibacterial Drugs: These medications are extremely helpful at times. If uncontrolled or partially controlled nasal allergy and asthma exist for any length of time, suppurative sinusitis and pulmonary infection will surely supervene as the result of edema and stasis of the mucous membranes. An acute attack of sinusitis and pneumonitis, responding to the anti-allergic regime and antibacterial drugs, will not become chronic. Antibacterial drugs must be given in adequate amounts over a prolonged period of time.

Irradiation: X-ray irradiation of the mucous membranes of the sinuses, in low grade, subacute, and chronic sinusitis, is recommended in certain cases. This has been, in my experience, a more useful form of therapy in children than in adults. It is administered to those patients who have not responded to conservative treatment and who do not present clear enough indication for surgery. The good results from irradiation therapy are often only temporary.

Miscellaneous Drugs: This group has been discussed by a previous speaker. I will not consider these drugs except to mention that I have obtained results in children with mild to moderate nasal allergy and asthma, with the use of a calcium and iodine preparation, administered orally.

Now, let us go on to the next slide and chart dealing with the indications for sinus surgery in asthmatics.

Number one, pathologic changes in the sinuses

TREATMENT OF SINUSITIS
IN PATIENTS WITH ASTHMA

SLIDE 2
INDICATIONS FOR SINUS SURGERY
IN ASTHMATICS

1. Pathologic changes in the sinuses requiring surgical treatment upon their own merits.
2. Minor to moderate sinusitis—persistent asthma not responding to prolonged conservative treatment.
3. Cases in which minor nasal procedures have been of definite benefit, e.g., sinus lavage, polypectomies.

requiring surgical treatment on their own merits. This means that if sinusitis is present and surgery is necessary to obtain improvement, then it should be performed, regardless of the presence or absence of asthma. Of course, asthma, if present, is given full consideration.

Another group of cases encountered consists of those with a minor to moderate degree of sinusitis and, in addition, persistent asthma which has not responded to prolonged, conservative, or medical treatment. In some of these instances, the allergists and we, the otolaryngologists, believe that we are justified in advising rather extensive sinus and nasal surgery. This is especially true if it has been repeatedly shown that the patient has improved temporarily after such minor procedures as sinus lavage, displacement therapy, nasal cleansing, and polypectomy.

The selection of patients for nasal and sinus surgery is done very carefully, and only after a prolonged period of observation, measured in months or several years, by the allergist and otolaryngologist.

Some will want to know of the accomplishments of surgery upon the patients with nasal allergy and asthma. As the result of our own experience here and of those elsewhere, I have accumulated certain figures that may be of interest.

Following minor rhinological surgical procedures in asthmatics, a few cures and improvement in perhaps 75 per cent of the patients may be anticipated.

In the cases of major or more extensive surgery, a fair estimate will be 50 per cent improvement in the asthma and rhinosinusitis. The asthma will not be improved in 50 per cent of the patients treated medically and by additional extensive surgery. It is also estimated that in this group of 50 per cent showing no improvement, there is the possibility that the asthma may be aggravated in 18 to 20 per cent.

Another figure which is of more interest to the rhinologist is that in approximately 65 per cent of the cases, the nasal and sinus condition is improved, disregarding the influence, favorable or unfavorable,

upon the asthma. In 30 per cent, the results of surgery upon the nose and sinuses will be indifferent; and in 5 per cent, the condition of the nose and sinuses will be aggravated.

The above figures are useful in any discussion with the patient and referring physician, in regard to contemplated rhinological surgery, with or without asthma.

In order that one may be somewhat familiar with the rhinological surgical treatment of these conditions, consideration will be given these procedures briefly in Slide 3.

TREATMENT OF SINUSITIS
IN PATIENTS WITH ASTHMA

SLIDE 3
NASAL & SINUS SURGERY

I. Minor—single or combinations.

Lavage	Middle Turbinectomy
Polypectomy	Submucous Resection
Antrostomy	Inj. Sclerosing Agents

II. Major—single or combinations.

Ethmoid—Exenteration
Intranasal approach
External approach
Antra —Removal of Lining Membrane
External approach
Frontal —Removal of Lining Membrane
External approach
Sphenoid—Partial Removal of Membrane–Window
Internal approach

Minor procedures, single or in combinations, such as lavage of the antra and, less often, the frontal and sphenoid sinuses, are helpful therapeutically and diagnostically. The relief of nasal and sinus obstruction, by submucous resection and polypectomies, is well recognized by all as being beneficial. Windows into the antral cavities, and, occasionally, the sphenoids, will do much to improve drainage and diminish the pressure from and within these areas. Middle turbinectomies, once so popular, have justifiably given way to a more conservative method of reducing the size of these structures—submucous resection of the turbinate. The use of sclerosing agents and the cautery upon hypertrophied mucous membrane and polyps has not had an enthusiastic following here in our group, but has been carried out with favorable results by some rhinologists elsewhere.

Major procedures, usually in combinations, will be commented upon briefly.

The cells of the ethmoid sinuses may be completely and safely removed, with or without a middle turbinectomy, by the external approach. A less complete operation may be performed by the intranasal method.

The diseased mucous membranes of the antral cavities are easily removed by the intraoral approach, through the anterior wall of the sinuses. Operations upon the ethmoid and maxillary sinuses are uniformly successful.

Some difficulty is encountered in obtaining a good result after removal of the diseased membrane, in the event of frontal sinusitis. Retaining the patency of the nasofrontal duct is the difficult problem here.

As a rule, all that one can accomplish, as far as the sphenoid sinus is concerned, is improved drainage by a window procedure in the anterior wall, because of the anatomical inaccessibility of these cavities.

In exploring the sinuses, one looks for and removes only that tissue which appears to be so diseased that the possibility of recovery, even with improved drainage, is impossible. Islands of only slightly diseased tissue may be left *in situ*, to aid in the regeneration and relining of the cavity. At best, the lining that is replaced by nature cannot approach a normal one.

TREATMENT OF SINUSITIS
IN PATIENTS WITH ASTHMA

SLIDE 4
LYMPHOID TISSUE — PHARYNX

1. Surgical Removal
2. Irradiation
3. Actual Cautery

In Chart or Slide 4, mention is made of lymphoid tissue in the pharynx. Indirectly, this tissue must be considered in the treatment of asthma complicated by infection in the sinuses, because it has a definite effect upon the development and progress of sinusitis. This fact is particularly true in children. It is desirable in certain cases, in order to influence favorably the course of sinusitis, that as much of this tissue be removed as possible. This is accomplished by surgery, x-ray, and radium irradiation, and sometimes by use of the actual cautery.

In conclusion, a few comments are made with reference to the otolaryngologist's approach to the problems of the treatment of sinusitis and asthma. The methods used here at the University of Virginia Hospital are briefly outlined.

The Treatment of Atopic Asthma

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Charlottesville, Virginia

THIS discussion is on the specific treatment of asthma that is due to sensitivity to foods, inhalants, and other allergens. At the moment we are not interested in symptomatic treatment. We are not interested in asthma due to infection, nor are we interested in asthma due to nervous tension.

Before treatment, it is imperative that we recognize the specific allergens that are important. This can be done by means of a well taken history, by well done skin tests properly interpreted, by use of trial elimination diets, and by trial periods of avoidance of suspected inhalants. When allergens are identified, there are two effective methods of treatment. First, one can avoid the important allergen. Or second, one can desensitize the patient.

As a rule, atopy is easier to control than infection or nervous tension. It should be possible in most cases to recognize all the important allergens and either remove them from the patient's environment or decrease his sensitivity to them so that they will no longer give severe symptoms. When possible allergens should be avoided.

Let us consider foods first. In general, foods are not a very important cause of asthma, especially in adults. However, food allergy is not uncommon in children. It is occasionally important in adults. It should always be looked for. Suspected foods should be avoided completely for a trial period. They should be re-introduced one at a time every few days after a period of trial avoidance. Those that consistently cause symptoms should be avoided indefinitely. Some allergenic foods can be eaten occasionally or in small quantity without producing symptoms. Others may be eaten cooked when they produce symptoms if eaten raw.

Inhalants are very common causes of atopic asthma. As a rule they are fairly easy to recognize. Whenever possible, important inhalants should be removed from the environment of the patient. If it is not possible to remove them entirely, then contact should be kept at a minimum. There are a number of inhalants. Perhaps the most important and the commonest one is ordinary house dust. One may get into dust at home or at work. The home dust can be partly controlled by using dustproof mattress and pillow covers, or by using a foam rubber mattress and pillow, and by removing upholstered furniture, draperies, rugs, curtains, and chenille bedspreads from one's bedroom. Patients who are sensitive to dust should avoid barns, mills, thrashing machines, and other dusty occupations. Cosmetics should be avoided or non-allergic cosmetics used. Mold spores are important inhalant allergens. One can avoid them by removing moldy furniture, by drying up basements, by avoiding rotting leaves and other rotting vegetation, and, in general, staying away from moldy places. Molds are sometimes very difficult to get rid of, particularly if the patient has an old and damp house. Epidermals are also important inhalant allergens. Feathers are most important for most people. They can be avoided by using foam rubber pillows or by putting a dustproof cover on one's pillow. Other epidermal inhalants should be kept out of the environment of patients having asthma or hay fever if possible. Dogs, cats, and other pets should be kept out of the house. Pollens are important seasonal allergens. Unless one can leave home during the pollen season, avoidance is not very successful. Air-conditioning is fine if the patient can stay in the air-conditioned room. There are not many patients, however, who can go into an air-conditioned room and stay there. Insect sprays and either tobacco smoke or other smoke seem to be allergens at times, but they are more often non-specific irritants.

When it is not possible to avoid inhalant allergens that cause important symptoms, an attempt at desensitization should be made. I call your attention to two things in that statement. First, desensitize people only when they cannot successfully avoid the allergen. Second, only attempt desensitization to inhalants that cause important symptoms.

In 1910, Dr. Noon, in London, began injecting minute amounts of a saline extract of grass pollen into individuals who had early summer hay fever. He demonstrated that the sensitivity of these patients to grass pollen would be decreased as demonstrated by conjunctival tests. Interestingly enough, when Dr. Noon made his first report, none of his patients had been through a pollen season. The only thing he demonstrated was that the conjunctival test was less active. He hoped that these patients would

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IN PATIENTS WITH ASTHMA

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Some difficulty is encountered in obtaining a good result after removal of the diseased membrane, in the event of frontal sinusitis. Retaining the patency of the nasofrontal duct is the difficult problem here.

As a rule, all that one can accomplish, as far as the sphenoid sinus is concerned, is improved drainage by a window procedure in the anterior wall, because of the anatomical inaccessibility of these cavities.

In exploring the sinuses, one looks for and removes only that tissue which appears to be so diseased that the possibility of recovery, even with improved drainage, is impossible. Islands of only slightly diseased tissue may be left *in situ*, to aid in the regeneration and relining of the cavity. At best, the lining that is replaced by nature cannot approach a normal one.

TREATMENT OF SINUSITIS
IN PATIENTS WITH ASTHMA

SLIDE 4
LYMPHOID TISSUE — PHARYNX

1. Surgical Removal
2. Irradiation
3. Actual Cautery

In Chart or Slide 4, mention is made of lymphoid tissue in the pharynx. Indirectly, this tissue must be considered in the treatment of asthma complicated by infection in the sinuses, because it has a definite effect upon the development and progress of sinusitis. This fact is particularly true in children. It is desirable in certain cases, in order to influence favorably the course of sinusitis, that as much of this tissue be removed as possible. This is accomplished by surgery, x-ray, and radium irradiation, and sometimes by use of the actual cautery.

In conclusion, a few comments are made with reference to the otolaryngologist's approach to the problems of the treatment of sinusitis and asthma. The methods used here at the University of Virginia Hospital are briefly outlined.

The Treatment of Atopic Asthma

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THIS discussion is on the specific treatment of asthma that is due to sensitivity to foods, inhalants, and other allergens. At the moment we are not interested in symptomatic treatment. We are not interested in asthma due to infection, nor are we interested in asthma due to nervous tension.

Before treatment, it is imperative that we recognize the specific allergens that are important. This can be done by means of a well taken history, by well done skin tests properly interpreted, by use of trial elimination diets, and by trial periods of avoidance of suspected inhalants. When allergens are identified, there are two effective methods of treatment. First, one can avoid the important allergen. Or second, one can desensitize the patient.

As a rule, atopy is easier to control than infection or nervous tension. It should be possible in most cases to recognize all the important allergens and either remove them from the patient's environment or decrease his sensitivity to them so that they will no longer give severe symptoms. When possible allergens should be avoided.

Let us consider foods first. In general, foods are not a very important cause of asthma, especially in adults. However, food allergy is not uncommon in children. It is occasionally important in adults. It should always be looked for. Suspected foods should be avoided completely for a trial period. They should be re-introduced one at a time every few days after a period of trial avoidance. Those that consistently cause symptoms should be avoided indefinitely. Some allergenic foods can be eaten occasionally or in small quantity without producing symptoms. Others may be eaten cooked when they produce symptoms if eaten raw.

Inhalants are very common causes of atopic asthma. As a rule they are fairly easy to recognize. Whenever possible, important inhalants should be removed from the environment of the patient. If it is not possible to remove them entirely, then contact should be kept at a minimum. There are a number of inhalants. Perhaps the most important and the commonest one is ordinary house dust. One may get into dust at home or at work. The home dust can be partly controlled by using dustproof mattress and pillow covers, or by using a foam rubber mattress and pillow, and by removing upholstered furniture, draperies, rugs, curtains, and chenille bedspreads from one's bedroom. Patients who are sensitive to dust should avoid barns, mills, thrashing machines, and other dusty occupations. Cosmetics should be avoided or non-allergic cosmetics used. Mold spores are important inhalant allergens. One can avoid them by removing moldy furniture, by drying up basements, by avoiding rotting leaves and other rotting vegetation, and, in general, staying away from moldy places. Molds are sometimes very difficult to get rid of, particularly if the patient has an old and damp house. Epidermals are also important inhalant allergens. Feathers are most important for most people. They can be avoided by using foam rubber pillows or by putting a dustproof cover on one's pillow. Other epidermal inhalants should be kept out of the environment of patients having asthma or hay fever if possible. Dogs, cats, and other pets should be kept out of the house. Pollens are important seasonal allergens. Unless one can leave home during the pollen season, avoidance is not very successful. Air-conditioning is fine if the patient can stay in the air-conditioned room. There are not many patients, however, who can go into an air-conditioned room and stay there. Insect sprays and either tobacco smoke or other smoke seem to be allergens at times, but they are more often non-specific irritants.

When it is not possible to avoid inhalant allergens that cause important symptoms, an attempt at desensitization should be made. I call your attention to two things in that statement. First, desensitize people only when they cannot successfully avoid the allergen. Second, only attempt desensitization to inhalants that cause important symptoms.

In 1910, Dr. Noon, in London, began injecting minute amounts of a saline extract of grass pollen into individuals who had early summer hay fever. He demonstrated that the sensitivity of these patients to grass pollen would be decreased as demonstrated by conjunctival tests. Interestingly enough, when Dr. Noon made his first report, none of his patients had been through a pollen season. The only thing he demonstrated was that the conjunctival test was less active. He hoped that these patients would

afford material for a later report. Evidently these patients did well because ever since then doctors have been injecting all sorts of things into people with hay fever and asthma. Soon after his original report, Dr. Noon died with pulmonary tuberculosis. His assistants carried on his work. His name still comes to us in the Noon Unit which is often used in designating pollen dosage.

Dr. Noon did demonstrate that some sensitive patients can be made less sensitive by repeated subcutaneous injections of minute but increasing amounts of a saline extract of appropriate allergens. This important principle still stands. Since his time innumerable reports have been written about desensitization. To the present, however, there is still little agreement on the details of this procedure. Authorities disagree on the methods of preparing extract, on the active ingredients, on the size of the dose, on the duration of treatment, on the interval between doses, the mechanisms involved in producing desensitization, and on the value of this form of treatment.

I shall not discuss the merits of any of the desensitization programs. I shall describe the procedure that we follow here. I am sure that every experienced allergist in the room has a different program that works better for him.

Let us consider first the patient who has uncomplicated hay fever and asthma from ragweed pollen. The following steps should be carried out:

1. Establish the diagnosis by history and by skin tests or conjunctival tests.

2. Begin preseasonal treatment as soon as the diagnosis is made.

3. Start with a dilution of the pollen extract that will probably not give a reaction. This varies from 1 part in 3,000 up to 1 part in several million.

4. The first dose of the first dilution should be 0.05 cc.

5. Increase the size of each dose by 0.1 cc.

6. When a dose of 0.5 cc. has been reached, increase the concentration of the extract tenfold and repeat the same procedure until a dilution of 1 part in 3.3 has been reached.

7. Increase the size of the dose of the 1 to 3.3 dilution until a dose of 1 cc. has been reached.

8. Give injections often enough to reach the desired maximum dose by August 15.

9. During the pollen season give 0.1 cc. every two weeks.

10. After the pollen season, start the perennial treatment by continuing the 0.1 cc. dose every two weeks.

11. Five months before the next season, start increasing the size of each dose by 0.1 cc. so as to reach a 1 cc. dose at the beginning of the pollen season.

This program will have to be altered many times to suit individual patients. Patients who start too late may not reach the ideal dose. Reactions may slow up the program. Patients who miss injections longer than 3 weeks should have the dose reduced. Patients who come in first during the pollen season or who develop symptoms in spite of preseasonal treatment should have co-seasonal treatment.

The same procedure is followed for other pollens, making allowance for the difference in dates. We try to reach the maximum dose for grass pollen on May 1, and the maximum dose for most tree pollens in March or April. When a patient gives reactions to several important pollens occurring at the same time, all may be included in the treatment mixture. We usually treat May and June patients with a mixture of grass and plantain pollen. We usually include a small amount of goldenrod, cocklebur, and corn pollen extract in the mixture for patients with August, and September symptoms.

Patients with perennial asthma may be treated with injections of the perennial inhalants. Common ones are house dust, feathers, other epidermals, fungi, kapok, orris, linters, and feeds. A treatment mixture for each patient is based on his history, environment, and skin tests. One should usually start with a dilution of 1 part in 600 to 1 part in 10,000. The injections are usually given twice a week. We usually work up to a 1:10 or a 1:5 dilution. One should give 0.1 to 0.3 cc. of this maximum concentration two times a week, one time a week, or once every two weeks depending upon the control of symptoms. A dose big enough should be given often enough to control symptoms. The interval between doses should not be greater than two or three weeks. If symptoms are controlled with dilute material, there is no need to increase the concentration further. For perennial inhalants there is no regular seasonal buildup. The frequency of the injections should be increased during periods of intensive exposure or exacerbation of symptoms. If symptoms should develop in a patient who had previously been controlled with dilute extract, the size of the dose should be increased.

Reactions to the extracts are an ever-present danger. They may be local, focal, or systemic. Severe

reactions are usually preceded by minor ones. When reactions occur, the dose should be held to the same size or reduced unless the reaction was a minor one. Never increase a dose that causes a reaction.

Reactions can usually be controlled by the administration of an antihistamine drug, adrenalin, aminophyllin, and a tourniquet above the site of the injection.

The results of treatment are variable. Patients with a clear uncomplicated clinical history and with corresponding skin reactions will nearly always receive very satisfactory relief from the use of injections if the proper antigens are used. As one begins to treat patients with less clear-cut indications, he will have fewer successes.

If one uses much skin testing and treatment material, it is more economical to make most of the extract from foods, dusts, epidermals, pollens, etc., as they are needed. If one has need for less material, he can buy the dried, powdered allergens and make his own extract. If he prefers, he can buy concentrated extract solutions or he can even buy various treatment mixtures already made up with proper dilutions for individual patients. The following are common commercial sources of allergy supplies:

1. Powdered Allergens:
   - Abbott Laboratories, North Chicago, Illinois.
   - C. G. Blatt and Co., 10810 E. 26th St., Independence, Missouri.
   - Graham Laboratories, Willow Lane, Rt. 7, Dallas 6, Texas.
   - Greer Drug Co., Lenoir, North Carolina.
   - Hollister-Stier, Wilkinsburg, Pa.; Spokane, Washington; and Los Angeles, California.
   - Lederle Laboratories, 30 Rockefeller Plaza, New York 20, New York.
   - Sharp & Sharp, 3402 Norton Ave., Everett, Washington.
   - Southwest Mold and Antigen Laboratories, 1009 N.E. 17th St., Oklahoma City 11, Oklahoma.
   - Stemen Laboratories, 1205 N.E. 18th St., Oklahoma City 11, Oklahoma, P. O. Box 6306.
2. Allergenic Extracts:
   - Abbott Laboratories, North Chicago, Illinois.
   - Barry Laboratories, Inc., 9100 Kercheval St., Detroit, Michigan.
   - Cutter Laboratories, 4th and Parker Sts., Berkeley 1, California.
   - Endo Products, Inc., Richmond Hill 18, New York. (Endo House Dust).
   - Hollister-Stier, Wilkinsburg, Pennsylvania; Spokane, Washington; Los Angeles, California.
   - Lederle Laboratories, 30 Rockefeller Plaza, New York 20, New York.
   - Parke-Davis & Co., Detroit, Michigan.
   - Wyeth, Inc., 1600 Arch St., Philadelphia 3, Pennsylvania.

What changes take place in the individual with asthma who has been treated with injections of appropriate antigens that make him less sensitive to these antigens? Dr. Mary Loveless and others have studied this problem intensively. The best explanation states that the injection of antigens leads to the formation of a heat stable or blocking antibody. This blocking antibody then inactivates the absorbed antigen, preventing a reaction between the antigen and the reagin and the production of symptoms.

The important steps in the specific treatment of atopic asthma are as follows:

1. Identify the specific allergen.

2. Avoid this allergen if possible.

3. If not possible, attempt to make the subject less sensitive to the specific allergen by injecting minute amounts of it subcutaneously.

4. During injection treatment, reactions are an ever-present danger.

5. Results will depend upon the care with which one selects his patients.

6. Treatment materials may be either made or bought.

7. Improvement seems to depend upon the production of blocking antibodies.

The Psychotherapeutic Approach to Asthma

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IT IS MY opinion that rarely, if ever, should we think of asthma as entirely psychogenic or attempt to treat it entirely by psychotherapy. By the same token, few, if any, cases of asthma are without a psychogenic element or to which psychotherapy could not be utilized with some benefit.

To quote Dr. Alexander at this point, "One of the most persistent errors in this field is the belief that if emotional etiology in a case has been established, somatic management becomes unnecessary and the patient can be turned over to a psychiatrist. This error is the reverse of the earlier misconception—namely: that if a patient has somatic symptoms, his case belongs exclusively to the domain of the physician outside of psychiatry. Psychotherapy aimed at the specific emotional factors of etiological significance is a long range project and must be coordinated with the rest of medical management. Above all, it must be correctly timed."

Fortunately, here at the University of Virginia Hospital, we have such a working arrangement. Essential agreement and mutual respect exists between the Department of Allergy and the Department of Psychiatry which has permitted a most pleasant and satisfactory relationship and the cooperative handling of many problem cases. The defects in this therapeutic approach lie mainly on the side of psychiatry and stem mainly from two sources: the present inadequacy of our knowledge and techniques, and the lack of sufficient time to meet the needs with which we are confronted.

The first step in the psychotherapeutic approach to any somatic condition is to establish a psychosomatic diagnosis. That is to establish to what extent, if any, emotional factors are playing an etiologic role. Obviously if the condition can be handled satisfactorily by relatively simple procedures, one would not resort to the difficult, time consuming and expensive measures which are still entailed in any definitive program of psychotherapy. In saying this, I am not forgetting that, in a sense, every procedure and every contact between the doctor and the patient has some psychotherapeutic significance. That is, it has meaning and an influence on the patient emotionally, for better or for worse. However, in speaking of the psychotherapy of asthma or any other psychosomatic disease, I mean more than what is implied by such terms as a good bedside manner or the art of medicine. I mean the conscious application of fairly well known principles and techniques to correct fairly definite pathology, psychopathology, which is in this case causing or contributing to a definite somatic dysfunction or disease. To avoid any possible misunderstanding, let me emphasize that when we use the term psychogenic asthma we do not mean imaginary asthma or hysterical dyspnea, hyperventilation or any of the other respiratory dysfunctions that may simulate asthma, although such phenomena undoubtedly exist.

The pathological physiological mechanisms of psychogenic asthma are the same as in primarily allergic asthma. It is only the pathogen or the stimulus that is different and it is to this etiologic source that we direct our attention in specific psychotherapy. Another aspect of the illness is also the concern of the psychotherapist; that is the meaning, the purpose, the value that the patient's symptoms may have for him in his total personality organization and in the constellation of his environmental setting. This may sound strange to the uninitiated that a person might find it useful, or necessary, or that he may prefer to be sick. The more sophisticated speak of the "secondary gains" sometimes in a ridiculing or condemning tone. We know from experience that somatic illness, especially the so-called psychosomatic illnesses, often do serve as a defense against severe personality disturbances such as depression or a paranoid psychosis. The psychotherapist then has to evaluate the total patient and his life situation, his assets and liabilities, strengths and weaknesses, in order to understand perhaps why he is sick in the first place, or what his illness has come to mean to him, what function it has come to serve and why he may with good cause be reluctant to give it up, and may, in fact, not give it up unless he finds a better, more effective or economic way of dealing with his problems in living.

The necessity for proper timing was referred to above. The acutely ill patient must of course first be treated symptomatically. In addition to the usual

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medical procedures, the psychotherapeutic measures at this point should be conservative, supportive, cautious, permissive, indulgent; not probing, uncovering, disturbing. After the storm is weathered, the attention may be gradually focused on the basic difficulties, with the expectation that as significant areas of conflict are brought to light, symptoms will recur or increase, and the speed of treatment will have to be regulated accordingly. In most cases, medical management and psychotherapy can and should be carried on simultaneously. The aim in this type of psychotherapy is to re-expose the patient to his conflicts, bring up the repressed, unconscious, forbidden memories, attitudes, feelings; help him to desensitize himself to them, help him to discard immature, inappropriate, reaction patterns; to accept himself and others more realistically, more rationally, and to learn to live more effectively, comfortably and productively. In asthma, the focus is usually on the unresolved, repressed, ambivalent dependency on the mother figure. This often includes a difficulty in handling emotions having to do with the desire for independence and, yet, a fear of it. Hence, attitudes such as aggression, self-assertiveness, hostility, give trouble. Also behavior associated with mature independent states, such as normal heterosexual activity, marriage, self-supporting work, etc.

But, obviously, I cannot here give a complete course in psychotherapy. The psychosomatic approach, which is necessary to good medical practice, emphasizes the necessity of not dividing the patient's problems into physical and mental but of treating them in totality. Yet, both the somatic and the psychotherapeutic techniques require specialists who cannot master both equally well. Teamwork in therapy and close cooperation between those from both disciplines is perhaps the best solution but not always practicable. Most doctors can become good psychotherapists, but he must be willing and able to learn the basic sciences of psychopathology and psychodynamics on which therapy is based.

A more efficient coordination of somatic and psychotherapeutic measures is one of the important challenges of modern medicine. It will require a more precise knowledge of the relationship of constitutional, emotional, and physiological factors in the causation of disease, and the development of improved, simplified and more easily learned techniques.

A fairly typical case seen recently for a follow-up visit may serve to illustrate some of the problems in treatment as well as some of the factors that are commonly found to be of etiologic significance. This case particularly well illustrates the team work approach we frequently use here.

Mrs. H. is a 49 year old white married woman, the mother of six children. She was admitted to the hospital on the Allergy Service in June, 1955, in an asthmatic spell that had persisted for about 2 weeks and had resisted all the usual medical measures. Here first asthmatic symptoms had begun only six months previously apparently precipitated by a cold and bronchitis. She had had a tendency to wheezing respiration since, with intermittent exacerbations of rather severe typical attacks of bronchial asthma. Physical examination revealed a moderately obese middle aged woman in respiratory distress. She appeared tense and rather depressed. There was an abdominal scar in R.U.Q. where gall bladder had been removed about one year previously. Otherwise physical and routine laboratory examinations were negative, except for typical physical signs of bronchial asthma.

The customary procedures for relief of symptoms were instituted and the allergic work-up begun. Because there were indications of psychogenic factors, we were asked to see her in consultation on the second hospital day. As she was still rather sick, we began by chatting with her quite casually, permitting her to talk at length about her symptoms. We, of course, told her from the first that we were psychiatrists and explained that we frequently saw patients with difficulties similar to hers which were felt to be in some way caused or aggravated by nervous factors. She had been previously prepared for our visit by the allergist and by her local doctors. She remarked that all the doctors had told her that her asthma was due mainly to her nerves and that she was nervous but didn't know why. The patient impressed one as being a very passive, dependent type, rather emotionally immature, anxious to please and cooperate, but also a person with a rather fierce sense of pride and an undercurrent of hostility. As we began to get the story and build up a working relationship with her in the ensuing days it was at first rather discouraging as she insisted on maintaining a very bland picture of herself, her environment, her personal relationships and her past life. There was a definite impression of subtle but firm resistance. After about a week of daily interviews in which very little of specific importance had been gathered but in which a fairly good rapport had been established and the patient was feeling better, we challenged her to the effect that she was holding out on us,

that she professed a desire to cooperate but actually she was not being fair, that we knew difficulties such as hers including the evident nervousness and depression always had causes which could be understood, etc. She became obviously angry and upset. We left her to think it over and that night she had a bad time of it, but the next day after much protestation of her wish to cooperate, she confessed that her oldest son on whom she had leaned a good deal had moved to New Jersey and had not been to visit her even since she had been sick because she had insisted that he not be notified. We called the son long distance and asked him to visit her mother in the hospital which he promptly did. To cut a long story short, we had already gathered from her that she was a mother who was actually quite dependent emotionally on her children. She had boasted a good bit about what good children she had and how devoted they were to her, how hard she worked for them, and how she could never deny them anything. Subsequent interviews brought out that a sixteen year old daughter was inclined to be wild, rebellious, had stopped school and the patient had worried a great deal over her and was incidentally trying to control her and punish her by being sick with asthma. Then came a dramatic revelation with a great deal of emotion that she herself had been an illegitimate child, had had a very traumatic childhood and had vowed to spare her own children anything like what her own life had been. There had been very little mention of her husband except that he was a good man and would do anything in the world for her.

Finally it came out that he was 18 years older than she, that he had retired as a coal miner at the age of 65, two years ago, and just hung around the house, leaving the family—including four teen age and younger children and herself—with no income except his pension. He took little or no interest in the home or children and never wanted to take her out anywhere. She said that his health was good, that he had been a heavy drinker in the past, and when the children were young he was gone from the house most of the time. Now that she was able to get out, he just wanted to stay home.

Many of our interviews were rather stormy. We encouraged the patient to express her aggressions more openly and she learned that even after she had been difficult, disappointing or even insulting, we still came back. She stopped apologizing for everything and became less defensive. Her manner became more self-assured and dignified.

In her interview yesterday she was much more animated, and aggressive, smiled and laughed frequently, and said she had had no more spells of asthma since leaving the hospital except one lasting about a week and associated with a bad cold. She was still nervous and suffered some from insomnia. Obviously all her problems were not solved but she did seem to be a much more mature person and was essentially free of asthma.

Sound Waves to Ease Pain.

High frequency sound waves have been used to relieve the unbearable pain of patients suffering advanced malignant diseases.

Dr. P. A. Lindstrom, of the University of Pittsburgh School of Medicine, said ultrasonic radiation to seal off a small portion of the brain was used in 17 patients as a substitute for similar treatment previously undertaken by surgery. Results of the tests indicated that the technique also could replace this type of surgery in patients with some mental diseases, eliminating certain unfavorable side-effects of surgical lobotomy. There were no complications or ill effects from ultrasonic treatment, and studies of the brain showed no apparent tissue damage.

Ten of the 17 patients, who had been suffering excruciating, uncontrollable pain, had practically complete relief for weeks to months after treatment and were able to discontinue previously heavy narcotic medication. Four other patients felt decreased pain and were comfortable a good deal of the time with less noticeable and one case was a failure.

Dr. Lindstrom said a major advantage was that there was little apparent tissue damage in the brain as may occur in surgical lobotomy. He made his report in the October Archives of Neurology and Psychiatry, published by the American Medical Association.

Treatment of Infectious Asthma

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THE treatment of infectious asthma requires at least four major considerations. *One* is to look for and treat the causes of wheezing other than infection. Remember that wheezing in chronic asthma is apt to be due to allergic *and* infectious *and* other causes. This is discussed in the section on classification of causes of wheezing. *The second* is symptomatic treatment, which Dr. Blanton has discussed so nicely already. Don't forget that, while symptomatic remedies are apt to be less effective in infectious than in allergic asthma, they should be used assiduously until specific measures bring relief. *The third* is the use of antibacterial remedies. *The fourth* is immunization with bacterial antigens. The last two supply the topics for this discussion.

Infectious asthma is treated with the same antibacterial remedies, by the same routes and in the same doses which are effective in other respiratory infections. A few additional therapeutic procedures, which are especially useful in infectious asthma, will be discussed briefly.

Infectious asthma may be acute, subacute, or chronic. The acute attack is treated, like any other respiratory infection, with sulfonamides, penicillin, streptomycin, achromycin, erythromycin, terramycin and aureomycin. In this clinic the selection of these remedies is more practical than scientific. Our patients are started on elkosin immediately, one gram four times daily. Elkosin is cheap and has a very low incidence of intolerance. Other sulfonamides are probably just as effective. If cough and wheezing are not markedly reduced in 48-72 hours, achromycin, 0.25 gm. four times daily is given. If no improvement follows this in two or three days, a combination of at least 600,000 units of penicillin and one gram of streptomycin is given daily. Few patients with acute infectious asthma fail to respond to one or the other of these antibacterial remedies. The order in which these three remedies are given is entirely arbitrary. Injections of penicillin are given with increasing reluctance because it causes so many reactions, even fatal anaphylaxis.

Cultures and sensitivity tests should be made promptly in order that a specific antibacterial drug can be used if the first arbitrarily selected remedy is not effective. The reason for the prompt administration of these remedies is that respiratory infections frequently result in status asthmaticus before results of cultures and sensitivity tests can be obtained.

There are *two common errors* in the use of antibacterial drugs in acute infectious asthma. The *first* is failure to continue an effective drug until the infection is cured. One or two doses of penicillin or eight to ten achromycin pills frequently provide 50% to 75% relief of the attack, but a little residual asthma may persist for weeks or months. Whereas, if the effective drug is continued for five to ten days the attack is usually cured. The *second* common error is to continue giving an antibacterial drug which fails to initiate significant relief within two or three days. Any drug which fails to give striking relief within three days should be discontinued and another substituted. The results of the cultures and sensitivity studies should be available by then.

Sinus infection is, perhaps, the commonest single cause of acute infectious asthma. Sinus infection must be recognized and treated promptly when present. Acute infectious asthma due to sinus infection is not apt to subside quickly unless this is done.

Unless other causes of wheezing are recognized and controlled and the infection is cured, residual cough and wheezing will persist in varying degree. This is *subacute infectious asthma*. This is treated like chronic infectious asthma.

Chronic infectious asthma is apt to have infectious *and* allergic *and* other components. Infected allergic respiratory membranes, as a rule, do not respond satisfactorily to simple anti-infection procedures. Treatment of the infection must, therefore, be supplemented by management of the other causes of wheezing.

Sputum is an important problem in the management of infectious asthma. It may be tenacious and difficult to raise. It seems responsible for much of the exhausting cough. Sputum seems to protect bacteria from antibacterial remedies at times. This problem is tackled from several angles. *Potassium iodide*, one gram four times daily, is given routinely if tolerated. The impression is strong, however, that potassium iodide is more effective in keeping sputum fluid than it is in liquefying sputum which is already tenacious and inspissated. There is a

great need for more effective methods by which this sticky material can be loosened and coughed up readily.

Aerosols are used effectively to apply topical antibacterial and mucosal shrinking agents. The formula used here is 25,000 units of penicillin and 50 mg. of streptomycin in 1.0 cc. of 1:1000 isuprel, administered every two hours. This should not be used, obviously, when the patient has reacted unfavorably to penicillin or streptomycin in the past. Other sympathomimetic compounds can be substituted for the isuprel. Perhaps bacitracin, gramicidin, polymyxin, achromycin, erythromycin, and other antibacterial drugs will become more popular for this purpose than they are now. An oxygen tank, a bedside compressor, or a home-made attachment for the vacuum cleaner will provide the stream of air with which to nebulize the drugs. The mouth should be rinsed thoroughly with glycerin water before and after each treatment, to minimize oral irritation. Cough and purulent sputum which are resistant to 5-10 days of this therapy require extensive bacteriological and other procedures.

Inhalation of 125,000 units of *Tryptar* once or twice daily for a few days is often of great aid in softening the tenacious sputum.

Bronchoscopic aspiration should be employed freely when the patient becomes critically ill, before the above measures can bring relief or when they fail. Remember that the lungs of patients who die from asthma invariably show extensive plugging of small bronchi by this thick tenacious sputum. Bronchoscopic aspiration may be life-saving.

Postural drainage has been disappointing. *Nasal polyps*, which are common in infectious asthma, should be excised promptly to remove the reflex component and to make room in the nose for sinus drainage.

Status asthmaticus, which means asthma which is resistant to conventional symptomatic therapy, *is seldom seen* in this clinic *except in infectious asthma. ACTH and cortisone* have almost, but not quite, removed the fear of status asthmaticus. Few cases fail to respond to these hormones. 100 mg. of cortisone by mouth, followed by 50 mg. every 4 hours, will relieve all but a small minority of status asthmaticus within 12-48 hours. 20 mg. of ACTH given in a slow intravenous glucose drip seems to initiate relief sooner than cortisone. Treatment in critically ill cases should be started with one ACTH infusion and the first dose of cortisone simultaneously. The presence of infection is not a contraindication to the unrestricted use of ACTH and cortisone in asthma, when appropriate antibacterial drugs are used concurrently. On the contrary, the hormones seem to potentiate the effectiveness of antibacterial drugs.

Treatment of Infectious Asthma by the Injection of Bacterial Antigens. This topic is difficult to discuss because nobody really knows what happens when an asthmatic is given bacterial antigens. Do we immunize? Desensitize specifically? Or non-specifically? Do we initiate the alarm reaction? Reams have been written, but in the final analysis, we are being empirical when we give bacterial antigens to asthmatics. They seem to help some infectious asthmatics, quite unpredictably, when other measures fail. The fact that they are used empirically does not contraindicate their use. We use them a lot.

Bacterial Antigens Should Not Be Given to Every Asthmatic. There is no reason to think they would help allergic, physical, cardiac, reflex, or obstructive asthma. Nor would they be expected to help the asthma from non-specific irritation, emphysema, fibrosis or cystic disease of the lung, unless complicated by bronchitis or bronchiectasis. Psychogenic asthma might be benefited, but distilled water would serve the same purpose. Asthma should not be treated with bacterial antigens simply because the patient does not give positive skin reactions to foods or inhalants nor because they do react to bacterial antigens. Bacterial antigens should not be used until more direct methods of treating the infection have been tried unsuccessfully.

Bacterial antigens should be given, empirically, when the infection is resistant to specific therapy and when there is evidence in the history, physical and X-ray examinations, that infection is *the* cause or *a* cause of wheezing in that patient.

Selection of the Bacterial Antigen. Having decided that the asthma is due to infection, what antigen should we use? Again we are empirical. Should stock or autogenous vaccines be used? Should soluble growth products (culture filtrates) be used? Alone? Or mixed with vaccines? There are voluble advocates of each of these choices.

Let's look at the problem. *A stock vaccine,* to be suitable for general use, must be a comprehensive mixture of respiratory and, perhaps gastro-intestinal organisms; although the gastro-intestinal ones are not often included. To get such a comprehensive stock vaccine, one would have to culture organisms from the wide variety of upper and lower respiratory

infections typical of the area from which the patient comes. That is hard to do.

The question of *autogenous vaccines* or filtrates is not as simple as it would seem. For example, the flora of the right antrum is often different from the left. These, in turn, may be different from the flora in the right and left ethmoids, or from the nasal or pharyngeal mucosae. At post-mortem in a recent case there were completely distinct flora in each of five infected sinuses. The relative importance of pathogenic *versus* saprophytic bacteria has never been settled. There is no evidence that the antigens of saprophytes are any less prone to sensitize than are those from pathogens.

To continue, the mere presence of an organism in a culture taken today does not in any way imply that the patient's asthma is from that organism. The patient who has asthma from an upper respiratory infection in November may, in March, have asthma from a lower respiratory tract infection due to completely different bacteria. So, an autogenous vaccine made in November would have little or no likelihood of influencing the asthma in March. The problem, as you see, is quite a complicated one.

The Role of Skin Tests in the Selection of Bacterial Antigens. Positive skin reactions to bacterial antigens are, if anything, less apt to be clinically significant than are those to foods or inhalants. We live in a constant barrage of bacterial antigens from birth to death. It is no wonder that most people react to some bacterial antigens. Bacteria are packaged antigens. Most organisms contain at least one polysaccharide and one nucleo-protein, probably more. The tubercle bacillus has about 10 known antigenic components. The streptococcus is perhaps equally as complex. The separate antigens of other organisms have not been studied as extensively as these two. Skin tests with vaccines elicit delayed reactions typically, presumably because the antigens are packaged and have to be "unwrapped" in order to produce reactions. The nucleo-protein fractions of bacteria elicit delayed reactions typically. On the other hand, polysaccharides tend to produce immediate wheal and flare reactions, indistinguishable from those due to dusts, pollens, and foods.

As with other antigens, skin tests to bacteria represent diagnostic leads, the significance of which is difficult to determine. In the case of bacteria, avoidance is not practical. However, (1) *clinical improvement* following (a) successful treatment of infection or (b) injections of the bacterial antigens, or (2) *an exacerbation* of symptoms after an overdose of the antigen are thought to be good evidence that the bacteria involved are likely causes of the asthma.

Practical Considerations. We pool the vaccines plus the filtrates from 72-hour broth cultures from a large number of organisms to make our stock bacterial antigen. This mixture is used routinely in those patients whom we treat with bacterial antigens. After about 4 months of injections, autogenous vaccines may be added when the response has not been satisfactory. This program is highly empirical. We have no evidence that it is superior to that employed by other allergists.

Commercial vaccines are, probably, just as effective. We would prefer them, for convenience sake, if they could be made to order inexpensively.

Treatment with bacterial antigens is guided by the same principles which Dr. Guerrant outlined for injection therapy of allergic asthma. Start with small, dilute doses which are increased twice a week. *A word of caution is necessary.* We start usually with a 1:1000 dilution of our stock bacterial antigens. Commercial vaccines should be similarly diluted. The initiation of treatment with undiluted commercial vaccines may be dangerous. Troublesome exacerbations of asthma have followed 1:10, 1:100, and rarely, 1:1000 dilutions of our stock antigen.

Desensitization vs. Immunization. What happens when we inject bacterial antigens into infected asthmatics? We don't know. It has been pointed out (1) that small subreacting doses seem to saturate antibodies and thereby prevent subsequent reactions to antigens, (2) that recovery from severe reactions leaves animals resistant to the specific antigen, (3) that active immunization protects against doses of antigen which would kill the sensitized animal. We don't know whether bacterial antigens benefit infectious asthma by one of these or by some unrecognized mechanism.

Regardless of the mechanism involved, a worthwhile percentage of *infected asthmatics seem to be benefited by injections of bacterial antigens.* Clinically, injections seem (1) to prevent bronchial reactions to antigens absorbed from infected sites in the upper respiratory tract, the bronchi, or less frequently, in other systems, (2) to ameliorate the effects of invasion of bacteria from the outside or infected areas, (3) to help to cure or suppress the infection.

Treatment with bacterial antigens is, at best, *a compromise* with the more desirable aim of curing

the infection. Unfortunately, this compromise is frequently necessary.

The *literature* on the treatment of asthma with bacterial antigens, while extensive, is pitifully inadequate. This conclusion is based on a review of about 30 reports on the results of treating a total of 1708 cases. About one-third of these were reported as being well, while another third were significantly improved. The rest were not improved. Empiricism was really rampant. For example, there was a complete lack of uniformity in the choice of antigens (soluble bacterial products, stock vs. autogenous vaccines), the size of the initial dose, the frequency of dosage, the size of increments, the total number of doses, the time required for improvement, the desirability of local reactions, etc. In spite of this, the results were about the same in most of the papers.

Miscellaneous Comments

Improvement following injections of bacterial antigens is usually slow, three to six months. Occasionally improvement starts promptly.

Over-Dosage with bacterial antigens is more difficult to recognize than with ordinary allergens. Reactions to dust or pollen injections are usually prompt, in minutes or a few hours. Aggravation of asthma from bacterial antigens is frequently delayed 18-24 hours and may last from one to several days. The reaction to one dose may last until the next. It is essential, therefore, that the patient be cautioned to report delayed accentuations of cough or wheezing. These focal reactions are usually, but not always, associated with delayed local reactions which are red and indurated, but need not itch. Mild accentuation of asthma symptoms is not undesirable if it is recognized and the dose is reduced. A 1:10, or 1:100 dilution of the reacting concentration sometimes brings prompt relief. Failure to recognize the delayed aggravation of asthma by the injections may result in intensification rather than the desired relief. If there is any doubt that injections are accentuating cough or wheezing, stop them for two weeks. Then observe the effect of repeating the last dose.

Once started, injections of bacterial antigens should be continued until the patient has been free of symptoms attributable to infection for at least a year. On the other hand, the injections should be stopped if there is no improvement within six months. There is no laboratory or clinical test which will tell us when a patient can stop injections with little chance of recurrence. Experimentally, desensitized animals become resensitized three weeks or less after desensitizing antigen injections are discontinued. Patients do not follow such a regular pattern. Experiments, underway in this laboratory, to test the hypothesis that desensitization with haptens will be more lasting, have been mentioned.

There is a *widespread belief that injections* of allergens or of bacterial antigens *should be interrupted* during intercurrent illnesses such as measles, whooping cough, pneumonia, mononucleosis, etc. *There is no known basis for this belief. No purpose is served* by stopping the injections. On the contrary, many instances of troublesome recurrences of asthma have been seen in patients whose asthma had been well controlled until effective allergen injections were discontinued during and after an intercurrent illness. No harm (or good) is done by reducing the dose and increasing the interval between injections, temporarily, to about two weeks, but *the injections should not be stopped.*

Summary

Infectious asthma is usually associated with other causes of wheezing. These should be looked for and treated. Infectious asthma does not respond as well, ordinarily, to symptomatic remedies as does allergic asthma.

Acute infectious asthma and acute infectious exacerbations of chronic asthma respond to treatment with the same antibacterial remedies which relieve non-asthmatic respiratory infections.

An effective antibacterial remedy should be continued until the acute attack or the exacerbation of a chronic infection is over, five to ten days in most instances. When antibacterial remedies are stopped too soon, some asthma persists and is easily flared up by non-specific aggravating factors such as temperature changes, fatigue, drafts, etc.

When one antibacterial remedy is not effective, in three or four days, try another. Ideally cultures should be made from nasal and bronchial discharges as soon as the diagnosis of infectious asthma is made. Sensitivity studies will then permit specific antibacterial treatment.

Antibacterial remedies can be applied topically as aerosols.

Thick tenacious sputum is frequently loosened by tryptar aerosol.

Infectious asthma is the usual cause of status asthmaticus, by which we mean asthma which is not relieved by symptomatic remedies.

ACTH, cortisone, and hydrocortisone can be used freely and effectively in infectious asthma if buffered

with appropriate antibacterial remedies.

The paranasal sinuses are infected in a high percentage of cases of infectious asthma. Sinus infection must be recognized and treated promptly when present.

Chronic infectious asthma or infectious asthma which recurs frequently is often benefited by the use of immunizing injections of bacterial antigens. The use of these antigens is empirical but is often effective. This problem is discussed in some detail.

Some Patients Don't Want to Be Reassured.

Sometimes the worst thing a doctor can do is to tell a patient he's perfectly well, a University of Virginia School of Medicine physician, Dr. Andrew D. Hart, has said. Some patients don't really want to be reassured. These are the patients with "cardiac neurosis," which may cause real symptoms of heart disease but may only be a substitute or cover-up for greater problems—such as tension and nervousness or fear of mental illness. This type of patient visits the doctor with complaints of heart symptoms, and is told firmly and reassuringly that he has no heart defect. The patient may then go to another—or more than one—doctor seeking another diagnosis. He may quote the first doctor as having said such things as "You have six months to live" or "I've never seen a worse heart than yours." The patient actually may believe this—was what the doctor said—since even normal memory is far from accurate, and nothing is so helpful as recollections to people who "dearly" need to believe something.

Further reassurance does not help. The patient appears to be pleased with good news, but gives himself away with such statements as "It's just got to be physical or it must be mental." He might admit he would be "greatly relieved" if anything tangible could be found, Dr. Hart said in the November 20 Journal of the American Medical Association.

The behavior of such patients suggests that the anxiety about their hearts is necessary to them. A tangible organic defect serves as a "lesser-evil" defense against greater, undisclosed fears.

"Most of these persons have been inwardly tense and high strung as far back as they can remember." "Nearly all admit to excessive nervousness and worry. Sometimes an insidious emotional depression has led to a stifling, 'heavy heart' sensation that is none the less real because it is impossible to describe."

Unrest, undermining of self-confidence, feelings of unreality, and dread of mental disease often result in a person's conviction that he has heart disease, and he becomes preoccupied with this notion. At this stage, a firm statement that there is no heart disease only makes the patient discontented. He doesn't know how to explain his feelings in the face of an excellent physical report.

Rather than an outright statement of good condition in such cases, Dr. Hart suggested that physicians should avoid a firm diagnosis and work for a closer relationship with the patient to find out what his deeper problem is. Some patients then will respond to treatment aimed at turning their energy and talents away from their concern about disease and toward outside interests and activities.

Management of Asthma in Children

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VARIOUS aspects of asthma have been considered today with the exception of those which are modified by the special conditions of childhood. At first one might think that these are minor differences, but we soon realize that not only is the patient smaller, he is less cooperative, his fluid and his nutritional requirements are different, and his abnormal and his normal chest examinations are entirely different from those of an adult. His susceptibility to respiratory infections is greater; his drug tolerance and emotional response to illness are not exactly like those of an adult. In addition we have more than the patient himself to treat. The patient's parents are deeply involved in the management of asthma of the child. The child is brought to the doctor, he does not come to the doctor by himself, and that changes the entire focus of approach to the problem, because the patient is there in an entirely, I started to say passive role, but he is there is a very actively resistant role. The mother complains that the child has so-and-so. If we ask the child: "Why are you here? What's bothering you?" he may look around and say, "I don't know." Sometimes the situation which the parents describe actually does not bother the child at all, but he is bothered by an entirely different group of complaints. All of these things make the management of asthma in children a slightly different problem from the management in an adult.

If we consider first, some of the special features of asthma in infants and children under 2 years of age, we find that making the diagnosis is some times extremely difficult and in many instances we may diagnose asthma only by excluding everything else that might reasonably resemble asthma. Sometimes these other things are eliminated as quickly as they come to mind by the history, the age of the patient, and the physical examination; in other instances, it may be necessary to go through quite a gamut of special diagnostic tests in order to be sure that one is actually dealing with allergic asthma.

Among the conditions which must be differentiated from asthma in young children is, first and foremost, the presence of a foreign body in the respiratory tract. Laryngeal disease, tracheal disease, atelectasis of the right upper or more often right middle lobe of the lung may simulate asthma. Cystic fibrosis of the pancreas is becoming more and more widely recognized, but it may present enough features that have an allergic flavor to be confused with allergy in young children. We have just seen such a patient who had been treated for four years as an allergic child, who does not have an allergy at all, but cystic fibrosis of the pancreas.

There are many children who wheeze because of tuberculous lymph nodes, and who present a picture very much like bronchial asthma. It is probably unnecessary to emphasize to this group, who are familiar with the work of Dr. Edwin Kendig in Richmond, the importance of tuberculin testing in children. We feel that a tuberculin test should invariably be applied to all children who are having respiratory difficulty and who are suspected of having asthma.

If we turn now to the complications of asthma in children, we find as an outstanding problem, their extreme susceptibility to upper and lower respiratory tract infections, and that their pattern of reaction to infection is quite different from that of an adult. In contrast to the fairly easily recognizable focus of chronic infection in an adult, a focus of infection in a child may be so minute that it may smolder along without being recognized between either acute re-infections or exacerbations of infections. It is very difficult to do the sort of study to determine the extent of infection in a child who is acutely ill with respiratory distress that one would do in an adult. Many of the tests and examinations indicated for a thorough scientific medical study have to be omitted or postponed in a child in favor of immediate treatment for infection on the basis of a presumptive working diagnosis.

In addition to foci of infection, and allergy elsewhere in the body, psychological factors in the family situation are of the greatest importance. Since they have been discussed so fully we will not do more than to reiterate that they may profoundly color both the family and child's reaction to the disease.

A series of slides are shown to illustrate some of the complications of asthma, atelectasis of the right

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upper lobe in a 5 year old during asthma, with re-expansion with steam and expectorants.

Chest x-rays of a 5 year old girl with very severe asthma whose plain films of the chest made during attacks showed only suggestive diffuse broncho-pneumonia with a little more infiltration in the right middle lobe, but whose bronchograms revealed congenital bronchiectasis of the right middle lobe.

Subcutaneous emphysema occurs as an infrequent complication of asthma and may cause marked dyspnea due to air in the mediastinum. This situation is illustrated by x-rays in two different children.

After one has tried to eliminate the possible complicating factors, and conditions which may simulate asthma, we are faced with the problem of how to treat an acute attack of asthma. We have outlined medications that are sometimes effective in treating acute asthma in children with the comment that the doses are pediatric doses which may not be the case in some of the regimens which you have. There are certain things which are important in trying to get a child out of an acute attack of asthma. The fluid balance: children get dehydrated very quickly and they often have a good deal of fever, so they need fluids and they often respond dramatically to steam. In our experience, the use of Alevaire is very helpful but it seems to be more efficient if the child is of an age where it can be put into a croup tent. Then one must be sure the croup tent is actually delivering as much oxygen and Alevaire as one would expect. Those are technical points, but they make a lot of difference and must be constantly checked. In older children who don't need oxygen and who don't like to be confined in a tent (and that is an important thing in children) a nebulizer attached to an oxygen tank or air compressor may be useful. Children don't like to have anything put over their mouths and it is often very difficult to get them to cooperate in inhalation therapy. The regimen of an expectorant followed by an inhalation of an antibiotic sounds very nice on paper. The whole procedure may take nearly an hour to complete even though the postural drainage is planned only for about 5 minutes. But in spite of the difficulties, these techniques often can apply to children if parents or attendants are persistent in their efforts to carry them out. Children are more apt to complain of the taste of something, the feeling on the tongue, and so on, and their objections limit the effectiveness of these medications a great deal. Our experience with Tryptar in children has been modified by the objections that Tryptar has been very irritating to the mucosa of the nose and the mouth. I have discussed this problem with Dr. Swineford, and we have started doing the things which he has suggested. We have had two children who have gone into acute bronchospasm after several days of the nasal and throat irritation from inhalation of Tryptar. So, because of the difficulty in getting the child to cooperate, we are limited in using some of the means which I am sure would be very helpful if we could apply them under ideal conditions in children.

It is not necessary for me to discuss the use of the adrenalin and aminophyllin after the previous speakers, except to say that children usually respond very beautifully to these drugs. The mistake which is often made with aminophyllin in a child who is being treated for the first time, is exceeding a dose of 5 mg. per pound of body weight. If this dose is exceeded before one knows how well a child tolerates the drug, the child may be very nauseated and may have a day or two of intractable vomiting as a result of the aminophyllin. This may be a harder thing to treat than the asthmatic attack. If they just vomited once and stopped, that would be just fine, as asthma often improves after vomiting. Some children object to the use of aminophyllin by suppositories. For best results I have found that mothers must be given explicit directions for the technique of inserting a suppository high into the rectum. If a child is constipated there is actually no point in using medication by suppository unless the rectum is emptied of stool. If the child is too ill, or the mother doesn't feel capable of giving a cleansing enema, then liquid instillation with an infant ear bulb-syringe will be more effective than a suppository.

The use of ACTH and cortisone in treating allergic children is a matter of much interest at present and we are all familiar with recommended dosage. I think many people don't realize that one can give children doses for full therapeutic effect, and the severity of the condition being treated, not the size of the child, determines the amount of hormone to be ordered. Because many of us aren't used to taking the blood pressures of these little children, we sometimes forget that children develop hypertension from ACTH and cortisone just as do adults. When starting steroids we may think that we are only going to use them for the safe period of maybe one or two weeks, but, oftentimes, as many of you have learned, you start and you can't let go. So it is very important to know what a child's blood pressure is and to follow that carefully while giving the hormones. The weight is a pretty good index of tolerance, al-

though I have seen cases who developed hypertension without having a great gain in weight. It is also important to watch the salt intake in these little children. An infant diet with a quart of milk a day or with a milk substitute, in which there is an even higher salt content, contains more than a gram of salt. There is also added salt in strained or junior baby food.

In planning a low salt or salt free diet for an infant or young child, mothers should be instructed to cook the proper foods at home without added salt. Oftentimes we have to suggest giving potassium chloride in addition, to keep the children's electrolytes in balance. Those aren't problems in children who don't need steroid hormones for long periods of time. As cortisone can be given by mouth, ordinarily it is much easier to use in children than ACTH. It is substitution and not stimulating therapy and must be decreased gradually to prevent adrenal insufficiency. If time allowed I would show you some slides comparing the cost of ACTH and cortisone therapy; intravenous ACTH is the cheapest way of using any of the hormones; I think it is about 20-40 cents a day, compared to considerably more than that for the oral treatment.

Non-specific irritants are very important in producing wheezing in young children, and I think those are very important in little babies. Babies who have ever wheezed for any reason, may wheeze on subsequent occasions when anything irritates their respiratory tract. An example is cough when exposed to cigarette smoke and other sorts of tobacco smoke. Many adults who smoke incessantly are very inconsiderate of the little child who has a respiratory difficulty. Another kind of smoke which is extremely irritating is that from wood, kerosene, fuel oil, or coal combustion. Some heaters which have poor vents and don't burn efficiently, produce fumes which are another non-specific thing that causes wheezing. This has recently been emphasized by E. A. Brown. It is important to have air in a house moist enough because dry air may act as a non-specific irritant. All of these things should be attended to as a routine part of the management of asthma in the very young child.

The antibiotic problem has been discussed very thoroughly just now. I think the mistake that many people make in treating infections in allergic children is to give inadequate dosage. A mother says "I took the baby to a doctor and he gave him a shot of penicillin and he was all right for 3-4 days," and so on. This "one shot" is partly the parents' fault and partly the doctor's fault. I think the doctor should always try to maintain the key position about when he's going to give medication and when he isn't going to give it. I am sure most doctors at present feel that one injection or a few tablets are probably not going to completely eradicate the infecting organism in the respiratory tract, but he may not emphasize this sufficiently to the parents. If we would tell the mother in the beginning, "Now mother, we think this infection may be getting stubborn, and we may need to treat it for more than one injection, and we may also need to change our medication during the course of the thing," then the mother would not go away from the doctor's office with the feeling, "Well, he gave one thing today and another thing when I went back, and I'm not sure that he knows just exactly what he is doing, and I'm going to go on down the street somewhere else." I think that if we let mothers of allergic children who have infections realize that such children need just as vigorous treatment to eradicate infections as an older person does, we may prevent the development of resistant organisms. We see more and more children with Pseudomonas, Proteus and E. coli respiratory infection, and as far as I know we have no entirely effective agent against these bacteria at the present time.

In the older age child, the treatment of acute symptoms is very much like that in young infants, but our second step is of course making an etiological diagnosis. The testing has to be modified just as in little children. Since scratch tests are easier to do than intradermals in most infants and children, besides their being so much safer than the intradermals, they are preferred for initial testing. How to do them? Sometimes one gets a good scratch test by taking the blunt end of an ordinary wooden applicator and turning it around on the skin. This gets away from the sight of the needle, and oftentimes is a very helpful thing in testing a child. The conjunctival test one would think would be a good one, but in my experience most children don't like to have anybody approach their eyes with anything like a dropper. If you can get cooperation I think this is a fine test. Dr. Biedermann in Cincinnati has devised an inhaler for testing; it looks very much like a Benzedrex inhaler, and one can insert into that a filter paper which is soaked with either a liquid allergen or a powdered allergen and the child sniffs it. There is of course a potential danger, that unless it is preceded by a scratch test to rule out the extremely sensitive child, one may encounter severe reactions.

What about the long range program for handling these children? There are just a few things, the afternoon in running along and I would just like to mention a few quickly, particularly breathing exercises. These are based on those devised by the Asthma Research Council—(Physical Exercise for Asthma).

This one is a little modification and the idea is to blow out on expiration and see how far the little light-weight ball can be blown across the flat surface. The description of these exercises can be found in a booklet published by the Chicago Medical Book Co., and costs 80 cents. The exercises are very simple and are the sort of thing that many children enjoy. Postural draining is, I think, another thing that is very helpful, if it can be done properly. A modification for children is to have them get on monkey bars or trapezes and to encourage them to hang by their knees on the trapeze and to do that for a definite time each day or as long a time as they are able. The bar can be hung in a doorway and most children will enjoy that sort of activity.

Another thing which is helpful in the way of exercises are those to increase tolerance to changes in temperature. These exercises were originally described by Duke in which he suggested immersing the hands in warm or cool water for a minute, each day making the temperatures of 1° warmer and 1° colder. Children can by this device develop a tolerance for temperature change, and as one is carrying out a definite program it helps the mother to get away from the idea that she must rush out with hat, coat, and over-shoes every time the child runs outside. Mothers soon learn that children do get sick from their exposure to sudden changes in temperature and that these exercises actually help to develop tolerance to temperature change. I think that they are a good addition to the management of asthma in children.

I can add nothing to what has already been said this morning on the last and a most important phase of asthma, the emotional aspect. In children they occur in a setting or are a reflection of problems that are present in the people who surround them. How rapidly one approaches their resolution depends upon how willing parents are to accept them as important. As I said in the beginning, in Pediatric Allergy one has more than just the child to treat—he has a whole family.

PR Tip of the Month

Accentuating the positive is an approach advocated by churchmen, community leaders, copywriters and insurance companies—to name a few. The medical profession would do well to adopt a positive attitude whenever possible: e.g., by substituting "professional liability" for the negative phrase "malpractice."

Insurance companies do not offer *death* insurance. They emphasize protection by calling it *life* insurance. In viewing the medical profession, nine out of ten patients seem to associate malpractice more closely with wrongdoing or guilt than with *protection for the doctor*. Doctors would find it to their advantage to correct that fallacy by using a positive term themselves.

Aspiration of Secretions in Asthma

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APART from diagnostic considerations, it has become increasingly obvious that now and then a severely ill asthmatic patient needs endotracheal aspiration. In the early stages of a long-drawn-out attack the bronchial secretions can, with some difficulty, be expelled by the patient. Over the course of several days epinephrine may cease to be of help and increasingly tenacious secretions become impossible to dislodge from the finer bronchi. They constitute multiple small foci of obstruction and are, in essence, foreign bodies. Even segmental or lobar bronchi become completely obstructed or impacted, adding atelectasis, with cyanosis and obstructive infection, to bronchospasm. Long before this time the patient needs help in evacuating his secretions. Auscultation of such a chest, in addition to what might be called the common "tea kettle" noises, will usually disclose rhonchi, suppression of breath sounds locally, or distant pectoriloquy. When it becomes obvious that aspiration is needed, one of two procedures should be chosen:

1. Nasotracheal Aspiration

The apparatus, which should be easily available in every hospital, consists of a suction pump and a number 16 F. plain urethral catheter. The catheter should be new and springy. If the tip is cut off obliquely, it will pick up secretions more readily. The patient sits on the edge of the bed with his head thrust slightly forward, his tongue out and held by his own hand, if possible; the catheter is inserted into the more open naris and passed into the nasopharynx. Once it has negotiated this curve, the configuration of the oropharynx focuses it at the glottis. As the patient takes a deep inspiration the catheter is quickly advanced and usually enters the trachea. If it does not, a slight modification of the position of the head will cause it to pass through the glottis. Phonation is now characteristically hoarse or impossible and a paroxysm of coughing is produced. Suction is not applied until the catheter is actually in the bronchus, since it will produce cyanosis if prolonged for more than four to six seconds. When the head is turned to the left, the advancing catheter enters the right bronchial tree; it is advanced, perhaps to the hilt, and retracted several times, being rotated meanwhile, as suction is applied. Secretions are sometimes so viscid that the catheter becomes clogged and must be removed and rinsed with water. To aspirate the other bronchus, the catheter is retracted to a point estimated to be above the carina, the head turned to the right and the catheter advanced again. It now usually enters the left bronchial tree which is cleansed in the same manner. Obviously there are many secretions far outside the range of this catheter, but the coughing that it produces may dislodge some of these finer obstructions and eventually mucoid tenacious secretions may be brought within range of the catheter tip. The procedure is quick, safe, simple and usually effective.

2. Bronchoscopic Aspiration

This procedure is a little more formidable than simple nasotracheal suction, but does not necessarily require the patient to be taken to the operating room. In an emergency, a bronchoscope, a battery box and a source of suction are all that are required and can be used effectively in the patient's room without moving him from his bed. Anesthesia is not essential.

For best results a safer and more effective procedure can be carried out in the operating room where some sort of anesthesia is available and oxygen and other resuscitative materials are at hand. If topical anesthesia is chosen, such as cocaine or Pontocaine, their intrinsic toxicity should be kept in mind. Moreover, in the asthmatic any material introduced into the pharynx or trachea, or simply the patient's apprehension occasioned by this act, may produce an alarming increase in the respiratory difficulty. One may need to give aminophylline or epinephrine intravenously. Our preference is to use deep ether anesthesia, with care during induction to assure adequate oxygenation by manual assistance. This avoids the possible toxic and bronchospastic effects of Pontocaine to which asthmatic patients seem peculiarly susceptible, and affords whatever beneficial effects ether may have on the bronchial musculature. When the plane of anesthesia is deep, the bronchoscope is introduced with oxygen flowing

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through the side-arm. A rapid inspection is made and the secretions aspirated and collected for culture and sensitivity studies. Infrequently, some unusual situation such as an unsuspected tumor or bronchial stenosis is found. As the anesthesia begins to lighten and the cough reflex returns, normal saline in 20 c.c. increments may be instilled through the scope and aspirated while the patient is being turned by the assistants into positions calculated to expose the various lobar and segmental bronchi to lavage. Quite frequently an amazing amount of stratified, gelatinous material and fine bronchial casts will be obtained in the collecting tube. Sometimes this sequence will break up an attack completely, due to a combination of factors. Now and then the wheezing may be worse for a few hours, but in general 75% of the patients get moderate to marked relief.

Bronchoscopic findings are sometimes difficult to interpret. One group of endoscopists has reported that nearly 35% of their patients had bronchial stenosis, usually of the superior segmental bronchus of one of the lower lobes. This report, however, is unconfirmed and in our experience bronchial stenosis is uncommon. It should be remembered that the examination is carried out under foreshortened monocular vision, without a standard light intensity, under difficulties imposed by haste and poor exposure; moreover, it is probably not without a certain degree of subjectivity, so that the observations of two careful observers in the same patient within less than a week may be strikingly different. One common finding of interest is spasm of the major bronchial tree below the upper lobe orifices. It is unlikely that this is confined to the lower bronchial tree, but it is seen with difficulty in the upper lobe bronchi, since their orifices cannot be properly inspected directly without a telescope. The same phenomenon can often be found in emphysema and allergic bronchitis.

In summary, our present indications for therapeutic bronchoscopy in asthmatic patients are: The prevention of asphyxia, the termination of a chronic attack, the relief of atelectasis and, finally, on the rare occasions when strictures are found, their dilatation.

AMA Issues New Catalog of Health Booklets

For a healthier 1955, the AMA's Bureau of Health Education presents its new catalog of "Publications About Your Health." Listing hundreds of new pamphlets on personal and family health problems, copies of these booklets may be secured for distribution to your patients through AMA's Order Department.

Several intriguing new titles include—*For Safer Cycling* which describes a community program for teaching children safe bicycling rules; *Is He Ready for Kindergarten?* outlines the points parents should watch for in the physical, mental and emotional development of their pre-school children; *The New Contact and Corneal Lenses* discusses the new type lenses and the types of people who can safely wear them; *Joe's Nervous Breakdown* explains why and how breakdowns occur and what to do about them.

Write today to the Bureau for a copy of the new catalog.

Exhibit on Industrial Solvents.

Uses and abuses of common solvents found in and around the home and factory are pinpointed in a new exhibit—"Industrial Solvents"—prepared by AMA's Bureau of Exhibits in cooperation with the Council on Industrial Health. Particularly stressed are the hazards involved in using carbon tetrachloride and other solvents incorporated in paints, paint and varnish removers, and insecticides. As a precaution, the exhibit suggests adequate ventilation and proper labeling, and lists the various symptoms and accepted treatments. The exhibit—suitable for professional showings—may be secured from the Bureau of Exhibits.

Closing Panel on Asthma

WYNDHAM B. BLANTON, M.D., Presiding
(Drs. Swineford, Dees, Guerrant, Garnett, Fitz-Hugh, and Dr. Fletcher Woodward)

THIS group, seated about this table, is, as you readily recognize, composed of the same experts who have discussed, this morning and this afternoon, various important phases of asthma. They are now ready to receive and answer any and all questions which the audience wishes to put to them. This seems to be a particularly happy way to bring together any loose ends and to conclude this conference.

The first question indicates that someone in the audience feels that we are requiring too much of medical students in asking them to take the elaborate histories necessary in allergy. The questioner seems to doubt whether medical students are capable of handling such an intricate allergy questionnaire as has been distributed among us today, and wonders if the student's time had not better be spent in reading more about allergy in the text-books devoted to that subject. I should like to ask Dr. Swineford to address himself to this question.

DR. SWINEFORD: That brings out my paranoia. As stated in the introduction, asthma and hayfever affected 3,500,000 people in 1938. They were third on the list of recognized chronic diseases and were responsible for the loss of 21,900,000 working days that year. Asthma was diagnosed more often than any other important disease in this institution in a recent 5-year period.

I never have, and never will understand the complacency with which curriculum committees make it impossible for medical students to learn to take care of such a common group of diseases with the same assurance with which they take care of heart disease, diabetes, thyrotoxicosis, peptic ulcer, etc. The average medical student is taught more about Addison's disease, which he will see once or twice in a life-time, than he is about asthma which he will see every week or so. If anybody can tell me why a student shouldn't be taught how to deal effectively with these common complaints, I'd like to hear it. I have been waiting for even a poor explanation of this for over 20 years. By a strange coincidence one of our recent graduates was in the office complaining of just this problem the day this was typed.

DR. BLANTON: Another question, Dr. Swineford. What is your belief regarding the so-called allergic balance?

DR. SWINEFORD: I don't believe that I can answer that to everybody's satisfaction. Clinically, it seems that a combination of etiological factors is often necessary to change the allergic state of the individual from a subclinical to a clinical degree. For example, we see many people who on auscultation will have mild degrees of wheezing. They are perfectly comfortable, they don't know they are wheezing—they are in what we consider a subclinical state. They can be thrown into a paroxysm of asthma by non-immunological or by immunological means such as: changes in the weather; emotional tension; the inhalation of non-specific irritants such as fresh paint or cigar smoke or the fumes of meth crystals; the ingestion of a food or the inhalation of an allergen to which they are sensitive.

Dr. Eyermann, of St. Louis, found that a group of seasonal pollen cases were also clinically sensitive to some foods. When the next pollen season came around he put them on a diet from which these foods were eliminated and found that the majority of them no longer required pollen treatment. From that he developed the idea of the so-called *total allergic load* as an explanation for the change from the subclinical to the clinical state. Dr. Warren Vaughan called attention to what he called the *balanced allergic state* in which other things than the immunological mechanism determined the incidence and the severity of allergic symptoms.

DR. BLANTON: What is your attitude toward the role of heredity in asthma?

DR. SWINEFORD: That is a controversial subject. Until recently the idea seemed to be pretty well accepted that the tendency to become sensitive is inherited as a dominant characteristic. Dr. Dees, what do you think about this? I am sure you have given this a lot more thought than we have, because you get them while they're young.

DR. BLANTON: Will you enlarge on this question, Dr. Dees?

DR. DEES: I think it is very difficult to be sure from the literature what most people mean by a family history of allergy. It is impossible to make comparisons with one's own and reported cases

because relationships are not clearly stated in the literature. It seems to me from our histories that the percentage of close family history is rather small. If one includes distant relatives, perhaps in as loose a fashion as could be reported by the family, so that one would include grandparents, aunts, uncles, and cousins, then I believe one would find about 55% of the patients have allergic relatives. If one considers only parents, then I believe it is much less than that. The incidence of sibling allergy among my own patients has been relatively small, but Dr. Glasser and others have reported fairly high incidence of sibling inheritance of allergy. I think the question is still one that needs to be reviewed and reconsidered. Whether Dr. Ratner's idea that the inheritance of allergy is rather limited turns out to be true or not, I think it would be worthwhile to have more precise information on this point.

Dr. Blanton: What is the type of inheritance in allergy? Is allergy a dominant or recessive character?

Dr. Dees: If there are few siblings, then that might explain why we see a rather small number of fathers and mothers who are allergic who have allergic children, but, if we go back to the grandparents, we deal with a much larger group, with much higher incidence which fits in more with the Mendelian theory of inheritance. The exact method of inheritance of allergy is not known. It has been stated that allergy is inherited as a Mendelian dominant with poor penetration. I am certainly amazed in looking over the records to see that the knowledge of family diseases is so different in different families. Some folks can tell you the most minute details about what was inherited and what other members of the family had, and others don't know anything about their family backgrounds. This makes data on inheritance based on patient's statements very inaccurate. I think until we can get these records of family diseases from doctor's own records rather than from parent's stories that we won't know what's what.

Dr. Blanton: Do you know of any statistics where the numbers of allergic children from non-allergic families have been compared with those from allergic families?

Dr. Dees: Yes. Those are the sorts of figures that Dr. Ratner has presented in his study. But, again, let me point out that if one tries to take hospital records of 100 children, with, for example, broken legs, to see how much asthma or allergy is reported in their family background as it is recorded, the chances are that there would be practically none because the presenting problem is not allergy.

Yet, later, many of those children may turn up in an allergy office, at which time the emphasis is on whether there is allergy, and then somebody will inquire for such a record and an allergic family history. I think that much of this information is biased because of the bias of the parents who are telling the story and because of the ears of the person who listens to it.

Dr. Blanton: I have several more questions which should be directed to the pediatrician on the panel. The first question is: Do children outgrow asthma?

Dr. Dees: I am sure some children do outgrow asthma. I don't think anyone has any accurate figures as to how many actually do outgrow these troubles. Swedish authors, who don't have the problem of ragweed pollen sensitivity, have stated that some 50% of the children outgrow asthma. In this country, as far as I know, no one has ever followed a series of untreated children who had allergic manifestations in early childhood to their later childhood to find out exactly how many of them outgrow the disease, but I think the impression one gets from pediatricians depends entirely upon how allergically oriented the pediatrician is. There are two physicians on our staff, for example, who have offices side by side and practices made up of the same general type of patients and living in the same locality. One man says that the incidence of allergy in his pediatric practice is not more than 5%. The other man says that at least 40% of his practice has allergic manifestations at one time or the other. Now, I am sure that those figures are biased by the man's own viewpoint. We need some thorough study of the allergic child's natural history. I think it would be safe to say that some children do outgrow their allergy because they become less sensitive to things. Other children outgrow it simply because by natural causes they are able to avoid the things they are sensitive to, and I think we just have to leave the figures unanswered and say that some do.

Dr. Blanton: Does anyone else have a comment on that?

Dr. Garnett: I would just say that from a psychiatric standpoint we do see those that do outgrow it and we think that part of how that occurs is simply in the maturing process and also upon the fact that the child, as he grows up, frees himself from his

traumatic home situation, if he is sensitive to the same sort of things to which you are referring.

DR. BLANTON: There is a saying among allergists that infantile eczema eventuates in hay fever and later in life in asthma. Now, can you take the infant with eczema and prevent him from having asthma?

DR. DEE: I don't believe that we can at the present time. That's what we hope to do, but the only study that I know of in which children with eczema were followed into older childhood didn't show figures that had reduced the incidence of the expected asthma in those children, and the incidence of expected asthma in children who have severe eczema is about 50%. The asthma may not be severe, but about half the children at least will have asthma. We hope that we'll modify it by treating the eczema.

DR. BLANTON: Some allergists favor, instead of direct skin tests on children, passive transfer. What is your attitude towards this matter?

DR. DEES: I think that passive transfer tests, just like direct skin tests, are a useful tool. I think they are even harder to interpret than direct tests and I think that the direct testing is the preferable method, as a general technique. I think passive transfer tests are very helpful under certain circumstances when the child's skin, for example, is too broken out to test directly; when he is too ill to be tested directly; occasionally for convenience, although that ordinarily isn't an important thing. But I think that it should be used as an adjunct to the other types of tests rather than the one on which one relies exclusively. Many people would disagree with these ideas, but that is my feeling about it.

DR. BLANTON: One final question you can answer yes or no; in hyposensitizing children, do you regard the weight and height of the child?

DR. DEES: No.

DR. BLANTON: Why not?

DR. DEES: Just as with the use of cortisone and ACTH we don't determine the dose by the size of the child but rather by the severity of the condition we are treating, so it seems with the desensitization technique that one should be guided by the degree of sensitivity the child has rather than his age. Little children can tolerate, often need, and are benefited by, exactly the same size doses that adults have.

DR. BLANTON: Have you actually used the smaller doses?

DR. DEES: I think everyone who has treated children goes through a period of doing that initially because he thinks, well this is a tiny baby. I just don't dare give him what I would give an adult. And then he finally learns from experience that they can take as large doses as adults and sometimes even larger doses than some older children and adults.

DR. BLANTON: Here is a question about something that has been much in the public thought of late—tobacco. How does the panel feel about tobacco? Do you, Dr. Dees, have any ideas on tobacco?

DR. DEES: I think that tobacco smoke is a nonspecific irritant for a great many people and a great many children. I think that as far as children are concerned—and they get smoke second-hand—the use of a filter doesn't make any difference. It has been my experience that there are very few children who are actually allergically sensitive to tobacco, and only a very few patients report they are unable to work in tobacco because of its producing respiratory symptoms.

DR. BLANTON: Suppose now we go to the psychogenic factor in asthma, and we will ask Dr. Garnett, can you apply to children the same methods that you apply to adults?

DR. GARNETT: We use the same methods with the exception of the factor that Dr. Dees mentioned that you have to deal more with the parents, more with the situations, more with the manipulation of the environment of trying to relieve or protect the child from certain factors in his situation and less with the actual unconscious and intrapersonal factors that the adult has incorporated into his own personality. I do think that a fair percentage of asthma in children is psychogenic and has to be treated psychotherapeutically if you are going to get a good result. The approach is somewhat different for the same reasons and in the same way that the medical approach to the child is different.

DR. BLANTON: In your field, do you ever use shock therapy or psychoanalysis for asthmatics?

DR. GARNETT: Shock therapy as a symptomatic treatment is sometimes used and sometimes has a dramatic and perhaps even life-saving result. The close relationship between asthma in some people and a severe psychotic state such as the severe depression or paronoid state is quite remarkable and both of them seem to respond to shock treatment as far as the acute symptoms are concerned. I do not think that that is the definitive treatment; it does not actually change anything. As far as psychoanalysis is concerned, of course that may be the definitive treatment. It is applicable, however, in

a relatively small number of people for various reasons. What we do use or advocate in most cases is a modified form of psychoanalysis or psychotherapy directed at the specific psychological factors in the personality and in the situation.

DR. BLANTON: One other question, Dr. Garnett. Do you believe this abnormal dependence on the mother is the result of some faulty relationship between the mother and child, or is the manifestation a personality defect on the part of the subject?

DR. GARNETT: That is a very interesting question which we cannot answer with any great degree of certainty. I would say probably both. There probably are children born with a certain constitutional tendency to be more passive, more dependent as we call it, more oral in their fixation, and there are others who are born with more aggressiveness, more tendency to develop along independent lines more readily. However, the environmental factors—particularly the early relationships between the mother and the child, do have a very large determining factor on what happens to the child, granted his hereditary predisposition. I would say in answer to that that both play a part.

DR. SWINEFORD: Can you give us any idea as to why maternal rejection causes unequivocally bad asthma in some youngsters and in grown-ups, but not in the majority who have the same emotional background?

DR. GARNETT: I perhaps unconsciously wishfully avoided the whole problem of so-called selectivity of psychosomatic symptoms, as to why one person might develop asthma, another nothing at all, and another eczema or stomach symptoms. This of course is an extremely controversial problem still. There are many ideas, none of which can fully explain the situation. The oldest theory was perhaps the so-called "weakest link" theory, that is that the system or organ which was most vulnerable was the system through which these emotional factors might be expressed. A reversal of that has been elaborated very recently by Wolf of New York who feels that it isn't the weakest link but the strongest link—in other words, the organ or system which has the most significance to the person is the one that he may utilize in this defensive way against his emotional conflicts. Then there is the factor of pre-existing disease, of a child, for instance, who has had chronic croup or frequently recurring croup as a very young child, then anxiety may become associated with the breathing function and it becomes a mechanism by which he gains the concern and attention of his mother. This undoubtedly plays some part. I would say that the factor of suggestion seems to play some role—the fairly frequent occurrence in a history of a child or an adult for that matter, having observed or been exposed to someone else in his environment who had asthma or hysterical dyspnea or pneumonia or something of that sort that made a striking impression on them and suggested the use of the respiratory mechanism as a means of expressing or discharging emotions. Finally there is the symbolic significance of the breathing, the exaggeration, as I have said, of a normal physiological tendency in all of us when under certain strong emotions to do such things as catching the breath or holding the breath or to pant or breathe heavily. The psychosomatic aspect, as I indicated at the beginning of my paper, is often thought of as an exaggeration or an intensification of a normal physiological response. Perhaps to try to answer very specifically as to why some do and some don't I would say that it is often a combination of these various factors plus possible unknown constitutional factors that we have to hypothecate.

DR. BLANTON: Dr. Swineford, would you comment on group testing, using groups of extracts, versus the use of the individual allergens?

DR. SWINEFORD: Our routine here is to group extracts of botanically related foods and test with a mixture of these foods as recommended by Dr. Warren Vaughan a number of years ago. That is an expedient, whether it is adopted or not is a matter of choice of the person who is to do the skin tests. Although many fruits and vegetables are botanically related, the evidence that they are antigenically related has not been explored satisfactorily. It is simply one of the short-cuts employed to cut down on the total number of tests necessary to provide a fairly comprehensive test coverage.

DR. BLANTON: Dr. Swineford, one more question, how can you get a patient off of cortisone once you've got him on it?

DR. SWINEFORD: I've got one arthritic I would certainly like to get off it. I don't know any way except just to take him off gradually and see what happens. During the period of removal, of course, there are the dangers of hypoadrenalism. The period when people are on cortisone should be utilized to the fullest to attempt to lessen their need for it. Too often there seems no satisfactory alternative to maintenance on the smallest effective dose.

DR. BLANTON: Dr. Dees, some pediatricians recommend the routine use of antibiotics and sulfona-

mides in the treatment of premature infants. Will this practice sensitize children to these substances?

Dr. Dees: I think that is a very good possibility, but I think that if the need for them is there and it very often is in premature infants, one has to take that chance.

Dr. Blanton: Dr. Woodward, do you advocate the routine removal of tonsils in all cases of infectious asthma?

Dr. Woodward: By no means. I think seldom is tonsillectomy indicated, but if you think that infection of the tonsils, as such, should be corrected, to control asthma, it would assist a great deal in the control of the disease if you eradicate the infection in the tonsils. Incidentally, if the tonsils and adenoids are factors in the continuation of the progress of the disease, they should definitely be removed, but not as routine.

Dr. Blanton: Has it been your observation that the staphylococcus is often found in causative relation to asthma? What are its effects on the tissues of the human body?

Dr. Guerrant: The staphylococcus in my experience has usually been an organism that we could get rid of or the patient would die, and lung infection with it has not been important in chronic infections in my experience. I have read that the staphylococcus toxin does have a peculiarly damaging effect on tissues including lung tissues, but I have no personal observation on that.

Dr. Blanton: Dr. Guerrant recognizes the importance of mild or subclinical asthma in the eventual production of emphysema. Should we not therefore, be more vigorous in our treatment of this state earlier in the development of the disease?

Dr. Guerrant: I don't know. I don't know why people get emphysema. We see some people that have asthma, quite severe asthma, for years and years and they don't get disabling emphysema. We see other people who never have any appreciable asthma who get severe emphysema. So I couldn't answer that with any assurance that I was answering it correctly. I would say that asthma seems to be one of the things that causes emphysema and, therefore, we should treat and control all asthma that we possibly can whether it is minor or severe.

Dr. Blanton: I'll ask Dr. Guerrant if he thinks ammonium chloride should be contraindicated in emphysema.

Dr. Guerrant: If one happens to have respiratory acidosis secondary to emphysema, which is one of the fairly common complications, ammonium chloride is likely to make that person worse, not always. As an expectorant for the treatment of emphysema, I don't believe ammonium chloride is very effective. In treating a person with emphysema who has heart failure and in whom you want to use a diuretic, and you need ammonium chloride to help you, I would see no objection so long as the patient did not have important respiratory acidosis.

Dr. Blanton: Here is the final question: "Please comment on some of the possible difficulties of the use of oxygen in the therapy of asthma." It seems to me that I might answer that by saying that oxygen lack is not a frequent need in the asthmatic. Oxygen is a drying substance. In asthma with emphysema, oxygen may actually produce alarming symptoms—the cessation of breathing and death in coma. The respiratory center becomes very insensitive in asthma, and the employment of oxygen may be disastrous. Any further comments on oxygen in the treatment of asthma?

Dr. Guerrant: It looks as if Dr. Blanton has covered it. Oxygen is seldom necessary in asthma, and in asthma complicated by emphysema it is dangerous. There are times when one has to use it.

Dr. Swineford: Oxygen doesn't do asthma any good. It is a ritual which you can duplicate simply by putting the patient in a tent and running in some cool moist air or steam or anything else you want to use. Oxygen is expensive and, more important, it takes the doctor's attention away from the patient's need for more definitive treatment.

Dr. Blanton: And yet I think it might be said that there are very few people in this audience who don't use it. It is very commonly used.

Notes On Pulmonary Tuberculosis . . . .

Modern Therapy VII
The Need for Balance and Perspective

The discovery of anti-tuberculosis drugs and the perfecting of daringly ingenious surgical techniques applicable to pulmonary tuberculosis, have, within the past few years, virtually revolutionized attitudes and practices with respect to this disease. But the many instances of truly miraculous performance by modern therapy must not be allowed to completely obscure the continuing validity and worth of fundamental precepts.

While the full impact of specific medicinal therapy and modern surgery upon tuberculosis is yet to be evaluated, there is nothing to date to indicate that either of these extremely useful treatment aids can completely replace familiar time-tested measures for the vast majority of patients . Indeed new discoveries and therapeutic procedures should serve to accelerate existing programs, for only by these means can potentialities of each new weapon be fully exploited.

Surgery has obvious limitations.

Nor has it *yet* been proven that any new drug or combination of drugs is uniformly and *completely* curative, *even for the earliest of minimal lesions*, or for healing the residual active foci which almost without exception remain, following *any* type of surgical extirpation. Available drugs are bacterio*static* rather than bacteriocidal in character—their total number is few—the dosage range of each is narrow—and they do *not* always perform "miracles".

A patient's ability to heal pulmonary tuberculosis is still dependent primarily upon his inborn constitutional resistance to tuberculous infection or his innate capacity to develop an "acquired" resistance after birth, which, for practical purposes, amounts to the same thing.

In order that this native resistance may be fortified and encouraged to expand fully, at least to the degree required to meet prevailing needs of the patient, it has been almost invariably necessary for those with clinical disease, to curtail wasteful dissipation of energy. In other words, the patient has found it necessary to *conserve energy*—one of the fundamental laws of Nature for combating infection in the absence of bacteriocidal or other *completely* curative therapeutic agents or procedures.

To reduce energy consumption one must REST—horizontally, vertically, or slant-wise, depending upon degree of conservation required. Reference has been made (see Sept issue, Virginia Medical Monthly) to the fact that "anything that is not exercise is rest". As a corollary one might add that anything *short* of maximal possible energy out-put for a given individual, over a 24 hour period, represents just so much conservation of energy during that length of time. A man standing idly on a street corner clearly is conserving energy by comparison to the effort he would be called upon to exert should he find occasion to mount and vigorously pedal a bicycle—and so on through innumerable gradations—until a conservation regime has been reached which can be achieved only by completely flat round-the-clock bed-rest.

It is well known that innate capacity to resist tuberculosis varies widely in different individuals. Unfortunately, in the past, an exceptionally good natural resistance has not invariably been associated with proportionately greater speed and thoroughness of recovery—*quite the contrary!* For one thing, those with an *above average* resistance were frequently tardy in consulting a physician—they didn't get *sick* enough *soon* enough!

In other ways, an unusually good inherited resistance to tuberculosis can be as a "double-edged sword". When properly exploited (everything else being equal) it *can* cut over-all time required for treatment and spare the patient many sacrifices and much needless physical suffering. But when used carelessly it has been known, with its other "edge", to nullify almost completely the *best* of "good intentions". When and *because* the disease could be so easily "controlled", it was just that much harder for the relatively untutored patient of yesteryear to accept the rigors of serious and consistent *curative* therapy—with the result that treatment in "quantity sufficient for the purpose" was often interrupted or postponed, again and again. Ten or twenty years later the patient with exceptionally good natural resistance still had active disease, often farther advanced with persistently positive sputum, while his tuberculous friend, with *half* the resistance, who needed *twice* as much rest, *and took it*, was well.

Modern drug therapy and surgery add tremendously to the average potentially curable patient's chances of getting well, and the latter requires much less rest than did his counterpart of a decade ago—but unless he capitalizes upon his good fortune,

Prepared by the Virginia State Health Department.

instead of making it an excuse to "play" at taking the cure (in terms of systemic rest) he is apt—five or ten years from now—to find himself in the identical predicament faced by the patient just described. And most of his Tubercle Bacilli, by that time, may be drug resistant, for whatever that may bode for him and for his contacts.

He, in turn, may have a tuberculous friend who, because of *below* average resistance to tuberculosis, combined with a dangerous allergy to the major drugs, (prohibiting their administration) may require four or five or more times as much rest to heal an approximately equilavent amount of disease; *but if the latter takes* the additional required amount—*chances are* that within three to five years *he will be well—and—he will stay well!* Many, many patients with none too good resistance *have done this* in the past, before the days of modern therapy.

Sight must not be lost of the fact that while there has been a marked reduction in the average *amount* of total systemic rest required in treatment of pulmonary tuberculosis, when supplemented by modern treatment aids, there has been and will be *no decline whatever* in the *crucial* importance of such systemic rest as may be prescribed in lesser amount, *as long as adjuvants remain adjuvants!* This applies to hours per day as well as to over-all treatment span as measured in months or years.

True, modern surgery and/or drug therapy *do* save time; they can save a *great deal* of time; the *danger* lies in the patient's thinking that one or both will save even more time than they do.

One of the biggest problems in tuberculosis treatment has always been to persuade the patient to take enough systemic rest, or when indicated, to accept appropriate aids, at the *right time!* Almost without exception, patients can be persuaded to do the right things, *too late!* A person can readily be induced to take fifteen hours bed-rest (or the equivalent) when he needs eighteen; when eighteen is no longer enough, he can be coaxed to take twenty—but by then his requirements may be twenty-four; even the *least* tractable will, at long last, readily accept twenty-four hours a day flat in bed when this amount may no longer be sufficient.

The only difference between taking 60% of the amount of systemic rest required, as compared to 95%, in a given case, is that in the latter instance the patient *gets worse more slowly*—much like jumping from the fire into a frying pan!

Sub-clinically active tuberculosis must be recognized for the important entity that it is, and treatment must be prescribed *specifically* for the purpose of rendering sub-clinically active tuberculosis inactive, over and above what may have been required to eliminate all evidence of *clinically* demonstrable *active* disease. (See Sept. issue, Virginia Medical Monthly) In the past many needless so-called "relapses" have been caused by failure to bear this in mind.

Orientation of patients, their families and friends, is absolutely essential in any chronic disease where effective treatment is dependent, as is often true in tuberculosis, upon group cooperation from start to finish. To promptly achieve and lastingly maintain perspective and balance in any treatment program, *all* members of the team must be thoroughly orientated upon basic facts about tuberculosis, *as a disease*, and its *principles* of treatment.

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Does prolonged systemic rest, especially "bed-rest", inherently produce harmful circulatory, skeletal, and metabolic changes? Apparently not. One might cite, for example, millions of patient-hours spent in hundreds of sanatoria by thousands upon thousands of patients, including scores upon scores whose physical immobilization was virtually complete for long periods, who seem to have demonstrated clearly that such unavoidable (and sometimes extensive) physiological adjustments as must take place under these circumstances, usually are not serious enough to jeopardize success of treatment nor, with extremely rare exceptions, do they prove to be irreversible when clinical improvements make possible progressive liberalizations of program.

On the other hand one *can* state *without equivocation* that systemic rest can be *dangerous* therapy when taken *haphazardly!* When taken for long periods in amounts less than "quantity sufficient" for the purpose, systemic rest *can* be *lethal*, with or without surgery, in or outside a sanatorium.

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For a long time to come many, many newly diagnosed patients will require prompt sanatorium admission, as they have in the past. *For this there can be no substitute*, if they are to be offered the greatest possible assurance of complete and permanent cure. Unfortunately many, many essentially bed-fast patents are being cared for today *at home, who should be in sanatoria*, if they would benefit fully from all applicable forms of modern treatment at a time when anything short of access to total therapy (*including periodic surgical appraisal*) deliberately courts ultimate therapeutic disaster. Ap-

parently there are many who are not yet fully cognizant of what modern excisional surgery has to offer, particularly for other than far- or moderately-advanced pulmonary tuberculosis—results undreamed of even a few years ago—potentialities which no bed-fast patient can afford to ignore—by staying home.

Undoubtedly there are some patients who could be helped greatly by anti-tuberculosis drugs who are not receiving these medicinal agents. During the calendar year of 1953 approximately 244 persons succumbed to tuberculosis in Virginia, who, prior to death, had not been reported to the Health Department. How many of these were diagnosed and for how long before death, is not known; *more* important, however, is that *more* such cases *be* diagnosed, reported, and treated!

Needless to say, of virtually all of the above, who *could* have been identified prior to death, *most* should have been hospitalized at some time—*most* should have received drugs. Had *all* been diagnosed and received appropriate treatment—their birthright in this enlightened age—*many* would not have died! Approximately 252 cases died from tuberculosis during 1953 in Virginia *who had* previously been reported to Health Departments. How many of these deaths were due to belated, half-hearted or misdirected efforts to get well?

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During the next decade or more the fate of tuberculosis eradication programs throughout the country will hinge upon the wisdom and skill with which physicians diagnose, prescribe, *and persuade their patients to accept* well balanced treatment regimes, tailored to meet the needs of each individual patient and geared to see the vast majority through to *complete* and *permanent* cure.

Sanatorium building programs likewise hang in the balance. Will hundreds of *new* beds (not now available) be needed *tomorrow* for curable patients who are being *almost* cured *today?* Will patients who can be cured without sanatorium care, *be* cured? Will patients discharged from sanatoria *complete* their cure *as most of them should* upon return to places of residence? These are not rhetorical questions but rather of deep concern to everyone interested in helping to eradicate tuberculosis as a major health problem within the foreseeable future.

It goes without saying that enough sanatorium beds *must* be made available to provide institutional care for *all* of those in need.

While it would plainly be poor economy to construct, *in addition*, expensive new sanatorium beds or to hospitalize in existing institutions *selected* patients who *could* be treated successfully upon a non-sanatorium basis, it would be even more wasteful to treat, or to attempt to complete the treatment of these patients, outside a sanatorium unless reasonable assurance can be had that *local physician interest* and facilities will be such as to virtually guarantee *comparable end results!*

Tuberculin Test.

Up to the present time the tuberculin test has been used most extensively in children. Great emphasis has been placed on its value in detecting evidence of tuberculosis in the younger age groups. However, with the marked decrease in the prevalence of tuberculosis infection observed in recent years among our adult population, the test becomes one of our most valuable weapons for the detection and ultimate control of tuberculosis. H. D. Lees, M.D., J. School Health, Sept., 1954.

Prolongation of Endurance.

That data which witness modern medicine's triumphs, the statistics that reflect the declining death rates and the increase in life expectancy (at birth), when critically examined add up to the summation that modern medicine has in a large measure converted mortality into morbidity. Those whose lives it has helped to save in many instances face not an extension of existence in well-being and in health, but merely a prolongation of endurance. Those who by the skills of modern medical science have been saved from death all too often have been saved only to longer endure the burdens of a variety of illnesses, and to die at a later age. In the statistical tabulations such individuals appear to have gained years of welcome life, but these may be, and too often prove to be, years of painful travail, years of dependency, unproductive years which in the last analysis are social and individual liabilities rather than assets. Iago Galdston, M.D., "The Meaning of Social Medicine," Harvard University Press, 1954.

Mental Health . . . .

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Christmas at Our Hospitals and Training Schools

At this season of the year all over the Christian world individuals and organizations endeavor to see that no one is forgotten. Those, who through economic conditions, are unable to provide toys for their children and the traditional Christmas dinner, are provided for by many interested individuals, groups and organizations with the sincere hope that no one is forgotten.

Those of us who are responsible for the care of the patients in our mental hospitals and training schools see that no one is forgotten. Individual gifts, special entertainment and the traditional Christmas dinner are enjoyed by all.

Many months in advance plans are made for the holiday season. With money obtained from profits of the Canteens, money provided from Trust Funds and other sources, gifts are purchased and wrapped in gay and attractive packages for all patients, so that no one will be missed. There are many patients who have no relatives of friends to remember them but they have the pleasure of opening their gifts along with those who do receive gifts from others.

The activity in the hospital mail rooms reminds one of the activities in a post office with the stacks of packages to be sorted and distributed to the thousands of patients. Extra workers and often volunteers help with this work.

Wards are decorated and Christmas trees are placed at various places over the hospitals and Santa Claus makes his visit on Christmas morning to all sections of the hospitals.

For the past several years each hospital has sent to the patients, members of their families and the employees a Christmas greeting card which carries a message from the Governor of Virginia, the Commissioner of Mental Hygiene and Hospitals, and the Superintendent of the hospital or training school. This card lists the program for the season and the Christmas dinner menu.

It is a heart warming feeling to read the program planned and carried out by the various hospitals for the pleasure of the patients and employees. With the thought that these programs might be of interest to you, the reader, some of the highlights for each hospital were as follows:

Eastern State Hospital, Williamsburg—

The season's celebration started on December 10th and extended until January 5th. Special music programs during this period were provided by the Mathew Whaley Chorus and Band, the chorus and choir of the College of William and Mary, Fred Epps and his quartette, the Girl Scouts and the Brownie Troops and other groups. Volunteer groups from the community had teas for women's groups and coffee parties for men's groups. Bingo parties were held for mixed groups and a pageant "The Nativity" was given by the patients.

On the 22nd a Christmas dance was enjoyed by the patients with special music provided. For those who did not dance, movies were provided. A Christmas party for the staff also was enjoyed by the employees on this day.

Christmas day was started off by the delivery of the gifts to all patients, followed by Christmas communion service. At noon a delicious dinner of roast turkey, cranberry sauce, candied sweet potatoes, fresh green peas, relishes and hot rolls, topped off with ice cream and specially decorated cakes was served. The festivities continued during the week with ward parties.

Western State Hospital, Staunton—

The festivities began here on the 18th with the lighting of figures on the lawn and the singing of carols. On Monday and Tuesday a Christmas pageant was given in the auditorium—repeated several times so that all patients could be accommodated in the auditorium. On the 22nd special Catholic services were held in the Chapel. All during the day special Christmas parties were enjoyed in the auditorium, ending the day with carols on the lawn. On Thursday parties still continued in the auditorium at the new unit (located outside of Staunton) ending the day with carols.

On Christmas day the gifts were distributed by Santa Claus and a dinner of roast turkey, cranberry salad, buttered broccoli, relishes, hot rolls and poin-

Article prepared by EDNA M. LANTZ, *Statistician, Department of Mental Hygiene and Hospitals.*

settia ice cream with petit fours made a happy day for the patients.

On Sunday the usual religious services were conducted. On Monday a special communion service for Episcopal patients was conducted. The judging of decorations on the wards and a Christmas dance completed the days activities. The employees were entertained at a buffet luncheon on the 28th. Movies and dances for the patients were continued, with the special programs ending on December 31st.

Central State Hospital, Petersburg—

The celebration of the Christmas season started here on December 23rd and continued through New Years Day.

On December 23rd patients and employees working together decorated the wards and Christmas trees. Movies were the order of the day for the 24th. Christmas day was welcomed by the singing of carols on the grounds and on the wards. A pageant, "The Nativity", was presented by the patients in the auditorium. This was followed by the distribution of gifts. A special Christmas religious service was held in the Chapel.

The traditional roast turkey, along with cranberry sauce, green peas, potato salad, relishes, hot rolls and mince pie was served to the delight of all patients.

For Sunday, Monday and Tuesday religious services and movies provided most of the entertainment.

On December 29th a Christmas play and a minstrel show on the 30th added much to the holiday entertainment.

The employees were entertained at a dance on Friday. The festivities closed with a New Years service on January 1st in the Chapel.

Southwestern State Hospital, Marion—

This hospital opened the season's programs with a bingo party on December 17th. Seasonal services were held on Sunday. Monday, Tuesday and Wednesday activities consisted of decorating buildings and grounds, with movies and caroling as added interests.

On the ward the patients held their Christmas dance, which was followed by the employees' dance.

On Friday, special Christmas parties were the order of the day.

On Christmas day the gifts were distributed and the judging of the decorations of the wards was made. Their dinner consisted of roast turkey, cranberry sauce, buttered peas, candied sweet potatoes, relishes, hot rolls, ice cream and fruit cake.

On Sunday the Christmas religious service took place. Parties were held on Monday. The season's festivities closed with a New Years dance on Tuesday, December 28th.

Lynchburg Training School and Hospital, Colony—

The festivities for the holiday season started on December 1st with a dance. On December 8th a second dance was enjoyed.

On Wednesday, December 15th, a Christmas program was carried out in the school. A religious pageant appropriate for the Christmas season was given on Sunday the 19th and a Christmas dance on December 21st. Thursday a caroler's party was enjoyed. During the entire period until Christmas day, movies highlighted the program.

On Christmas day a party was held on each ward and Santa Claus distributed the gifts. Christmas dinner here consisted of roast turkey, cranberry sauce, mashed sweet potatoes, green peas, relishes, hot rolls, ice cream and fruit cake.

The holiday season closed with a New Years dance on December 28th.

Petersburg Training School and Hospital, Petersburg—

The Christmas celebration opened with a Christmas pageant and a dance on December 23rd. On Friday, games, lighting of the Christmas trees and carol singing made an enjoyable day for the children.

On Christmas day gifts were distributed by Santa Claus followed by a dinner of roast turkey, cranberry sauce, kale salad, baked sweet potatoes, relishes, hot rolls and fruit cake.

The festivities continued through New Years with bingo parties, dances, talent shows, competition games, climaxed by ward parties and a dance on New Years day.

The planning and carrying out of these Holiday festivities for approximately 13,500 patients was work in addition to the regular hospital routine. Many employees spent extra hours in making the season happy and enjoyable for these patients. Their compensation was giving pleasure to these people who were unable to enjoy the holiday season in their homes and communities. It was "a job well done".

Public Health . . . .

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

The State Health Department's Meat Inspection Service

During the last session of the General Assembly an enabling act was passed authorizing the State Health Department to set up a meat inspection service. This service is to be voluntary on the part of the packers and must be self-supporting. The service is to operate under regulations adopted by the State Board of Health which are not less than those under which the Federal Meat Inspection Service operates.

Many meat packers and processers in the State have indicated an interest and several are already under the State Service's supervision with more to follow as the program grows. This interest can be attributed to a desire on the part of the packer to assure his customer of a clean, wholesome product and to afford him access to large urban markets that require all meats to be inspected before sale.

It is well known that numerous infectious diseases of animals are contagious to humans. It can be readily seen that intimate contact with diseased animals or their flesh increases the possibilities of transmission taking place. Admittedly, thorough cooking eliminates this hazard to a great extent but many cuts are consumed in a rare state and are therefore not protected. Handling of raw meats may also play a part in spreading certain infections, notably brucellosis, as is indicated in studies among packing plant employees.

At about the turn of the century Sinclair Lewis's book "The Jungle," and its description of conditions in the packing plants around Chicago, prompted the public to demand of Congress a regulatory service covering meats moving in interstate commerce. Out of this has arisen the modern inspection service under the U.S. Department of Agriculture. However, only plants where twenty per cent of their products go interstate are eligible for this program and numerous smaller plants are left out. Several other states do have a state meat inspection service (e.g. California and Pennsylvania) but many packers operate with little or no inspection.

In Virginia some of the cities have maintained an inspection service which has been adequate. The thought was advanced that a central state agency would be able to conduct a broader and more uniform service; hence the program was established in the State Health Department.

The Virginia program is under the direction of a veterinarian with considerable experience in meat inspection. Lay meat sanitarians will be assigned to the various plants participating in the plan to supervise sanitation and routinely examine each carcass or product processed. Where slaughtering is carried on a veterinarian residing in the area will be assigned to conduct final post mortems on those animals found in questionable condition. The live animals are observed for signs of disease and all vital organs including the lymphatic system are scrutinized post mortem for gross pathology. When necessary, histopathological work is carried out to facilitate final decisions. Chemical and serological tests are conducted to determine the composition of various products. Nothing will be left to chance in assuring the consuming public of a wholesome end-product.

Most of the urban areas of the State, on the basis of this new program, are planning to establish requirements restricting the sale of meat in their jurisdiction to that inspected by the Federal Government, the State, or an inspection service approved by the State Health Department. This should go far towards promoting uniform standards for meat and meat products throughout Virginia.

Since the service must be self-supporting under the present act, a fee is charged against the participating plant on a per hour basis. This money is paid to the State and is used in furnishing the various auxiliary necessities, travel, and salaries. All personnel involved in an official capacity are State employees and are in no way subject to undue pressure from the plant operator. The regulations provide for the operators' right to appeal decisions to higher authority when legitimate grievances arise.

Each wholesale cut from a meat carcass and all packages of meat products processed under this inspection service bear the seal "Inspected and Passed, Virginia State Department of Health".

The service is available to any qualified packer in the State who indicates a willingness to cooperate under the regulations. After an application is received, a thorough inspection is made of the plant, facilities and surroundings. When inadequacies are

corrected, inspection is begun as soon as qualified personnel are available.

It is hoped that this service will do much to enhance the prestige of Virginia meat packers and assure the consuming public of another reliable source of clean, wholesome meat and meat products.

MONTHLY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	Dec. 1954	Dec. 1953	Jan.–Dec. 1954	Jan.–Dec. 1953
Brucellosis	1	2	44	58
Diphtheria	3	17	36	97
Infectious Hepatitis	148	258	3544	2551
Measles	207	123	23816	4928
Meningococcal Infections	15	20	105	193
Poliomyelitis	17	16	605	740
Rocky Mt. Spotted Fever	0	2	41	61
Streptococcal Infection (Including Scarlet Fever)	496	400	5061	5405
Tularemia	12	25	44	55
Typhoid Fever	10	2	68	54
Rabies in Animals	37	48	367	460

Antimalarial Drug in Blood Disease.

An antimalarial drug has been found useful in treating patients with a blood disease in which too many red cells are produced.

Dr. Raphael Isaacs, Chicago, said in the December 18 Journal of the American Medical Association that red blood cell counts which had risen above normal were cut back and held steady by pyrimethamine.

The drug, used to treat malaria, is known to act against folic acid, a natural vitamin which helps in the production of red blood cells. Pyrimethamine was used by Dr. Isaacs to treat patients with polycythemia vera, a type of anemia in which the bone marrow creates red blood cells faster than the body can use them. There appeared to be no ill effects from the drug, but that it probably should not be used during pregnancy. Improvements brought about by pyrimethamine included normalizing of the red cell count, cessation of headaches and joint pain, lowering of blood pressure in hypertensive patients, decrease in itching, and "a feeling of well being."

Eyestrain May Not Be Caused by Defects.

Eyestrain, and the bleary look that sometimes goes with it, may be a product of civilization and not of disease or defect.

An item in the November 20 Journal of the American Medical Association said that eye strain is considered to be mostly muscle fatigue, resulting not so much from total expenditure of energy as from constant shifting and changing adjustments in a "futile groping" after a perfect picture.

Biologically the eyes were adapted "for relatively simple purposes—to look for enemies and for food," a consultant said in answer to a query to the Journal. Although the conditions in which we live today are accepted as normal, the eyes may not have evolved enough to meet the demands of "unremitting close work imposed on them by a highly complex and artificial civilization."

Some eye-strain is due to the tiring of higher perceptual processes, such as interest and attention, and some is mental-emotional rather than biological.

"Many a patient with bitter complaints concerning eye-strain and difficulty in carrying out the day's activity is but manifesting his hostility to his work, his home, or other factors beyond his control."

Medico-Legal Notes . . . .

Marriage, Divorce and Mental Disorder

"The contract to marry is extremely simple. It does not require a high degree of intelligence to comprehend it." By this dictum, one court indicated that a man might be mentally incompetent to buy a crate of cheese while he could be mentally competent to marry. In laying down this rule, the judges were not being unromantic. They were recognizing one of the facts of life. That fact is: every child is familiar with the give and take implications of marriage. Even a five year old knows the standard American marriage pattern. Daddy is away all day earning the money and in return mother cares for the children, cleans the house and cooks the meal. Thus the obligations implicit in the marriage contract are simple: daily work in return for daily work. The average child can grasp it.

And it follows, therefore, they will not set aside a marriage on the theory that the spouse did not know his obligations—they will not annul such a marriage unless there is overwhelming evidence that the husband or wife was so deteriorated, so confused as to lack realization of even this fundamental obligation. And this, remember, is within the ken of a five-year old child.

Any contract may be set aside if it is shown that one of the parties did not know what he was doing. If a man is so defective mentally—or so confused, so deteriorated, so drunk, that he really did not know what it was all about when he said "I do", the courts will annul that marriage. But it is against public policy to annul marriages. So this step will not be lightly taken. The assumption is that when a pair of adults marry they know what they are getting into. Anyone who wants to challenge that assumption has to prove the lack of capacity. No matter what the clinical diagnosis, the incompetency is not assumed.

The doctor is sometimes asked to testify that the bride or groom did not know what he (she) was doing at the time the marriage was consecrated. This comes up particularly with marriages performed when one of the spouses was drunk. Sometimes the question is raised when the bride is a young teenager. Or one of the parties might be a mental defective. The criterion in each case is not the diagnosis, but rather the question of whether the party had wit enough to know what he was surrendering by marriage and to know what obligations he was assuming. The fact that he could not pay off on those obligations is not important at this stage. The only question is: did he know the obligations? If he did not, was this because of mental disorder or because of lack of sophistication? If because of mental disorder, the doctor can say that the partner lacked capacity to marry. This does not automatically mean annulment, however. Courts are loath to annul marriages and there are several legal criteria in addition to this medical one.

Sometimes an effort is made to annul a marriage because of fraudulent concealment of mental disorder. Now fraud can annul a marriage—but it is only that species of fraud which goes to the "essence" of the marriage. If a young man, while wooing a maid, says he is president of the company, earning $25,000 a year when, in truth, he is simply the hand who packs the frankfurters, earning only $1500 a year, this is, of course, a fraud. But the mi-led bride can no more have her single status restored than she can get back her virginity. For this fraud, say the courts, does not go to the "essence" of the marriage. Misrepresentation about one's skills, education, character, income, reputation, and ability to cook are simply part and parcel of the technic of courtship, and are no more "fraudulent" in this sense than the puffs one sees daily in television. They may be misleading, but the doctrine of *caveat emptor* applies in courtship as in advertising.

Actually, fradulent concealment of a mental disorder is almost a self-contradiction. Fraud implies *willful* deceit. But if the mental disorder is serious enough to wreck the marriage, it is serious enough to impair the person's accountability. If the fraud thus is not "willful", it is no fraud. In any event, the rule about dissolving marriage to an incompetent spouse is for the protection of the sick one, not for the protection of the healthy spouse.

If the disorder is a hereditary one, or if it is loathsome, crippling or contagious, then its *willful* concealment might be grounds for annulment. But even here, the healthy spouse would have to show

Contributed by HENRY A. DAVIDSON, M.D. DR. DAVIDSON, *author of the standard* Forensic Psychiatry (*Ronald Press 1953*), *is instructor in Legal Medicine at the Medical College of Virginia and Assistant Superintendent of the Essex County Hospital at Cedar-Grove, N. J.*

that his partner deliberately and knowingly concealed the disorder with the intent to lure the other into marriage.

With respect to divorce, the problem is different. In the first place, psychosis (insanity) is, in most states, a complete bar to divorce. Though a woman may be childless and only 20 years old, she is forever tied to her psychotic husband even though he is destined to spend the next 45 years in the back wards of a state hospital. She cannot sue for divorce, for divorce implies misconduct on the part of the defending spouse. And mental disease is not misconduct. There are a few states which allow some kind of judicial dissolution of marriage if the spouse is irrevocably psychotic. But in most states the doctrine is that the couple took each other "in sickness and in health till death do us part." And mental disease is sickness.

Willful refusal to engage in sexual intercourse is considered as "desertion" in most states. If desertion is a ground for divorce (and it usually is) then willful refusal to engage in copulation is a ground for divorce. The operating word here is "willful". Ordinarily a man does copulate if he can, so that the husband's failure to, or inability to copulate, is generally considered to be non-willful. It therefore is not a ground for divorce. In rare cases, it might be shown that it was willful. (As in one case where, because of his religion, the husband would not use contraceptives, but did not want children, so he willfully refused to engage in coitus with his wife, using a sterile mistress as his outlet. The wife could not prove the adultery, but she could prove the "desertion".) If the wife refuses to engage in, or submit to, sexual intercourse because of neurotic fears, because of prudishness, because of hysterical belief that sex is sin, or because of neurotic vaginismus, then her behavior is generally considered willful, and therefore is a form of desertion. Regardless of what the doctors say, most courts take the position that such a reaction on the part of the wife is a willful denial of the husband's "rights". The psychiatrist may consider such behavior by the wife as a form of illness. The judge is more likely to construe it as a deliberate evasion of a "duty". Most laws, it appears, are written by men.

Book Announcements . . . .

Hugh Roy Cullen. A Story of American Opportunity. By ED KILMAN and THEON WRIGHT. Illustrated by Nick Eggenhoffer. Prentice-Hall, Inc., New York. 1954. viii-376 pages. Cloth. Price $4.00.

Handbook of Emergency Toxicology. A Guide for the Identification, Diagnosis and Treatment of Poisoning. By SIDNEY KAYE, B.S., M.Sc., Toxicologist, Office of the Chief Medical Examiner, Commonwealth of Virginia; Associate Professor of Legal Medicine, Medical College of Virginia; etc. Charles C. Thomas, Springfield, Illinois. 1954. xviii-202 pages. Cloth. Price $5.75.

Review of Medical Microbiology. By ERNEST JAWETZ, Ph.D., M.D., Professor of Bacteriology and Lecturer in Medicine and Pediatrics, University of California; JOSEPH L. MELNICK, Ph.D., Professor of Epidemiology, Yale University School of Medicine; and EDWARD A. ADELBERG, Ph.D., Associate Professor of Bacteriology, University of California. Lange Medical Publishers, Los Altos, California. 1954. 360 pages. Illustrated. Price $4.50.

Handbook of Medical Treatment. Edited by MILTON J. CHATTON, M.D., SHELDON MARGEN, M.D., and HENRY D. BRAINERD, M.D., Fourth Edition. Lange Medical Publishers, Los Altos, California. 1954. 569 pages. Cloth. Price $3.00.

Ciba Foundation Symposium On the Kidney. Arranged jointly with the Renal Association. A. A. G. LEWIS, B.Sc., M.D., M.R.C.P., Editor for the Renal Foundation. G. E. W. WOLSTENHOLME, O.B.E., M.A., M.B., B.Ch., Editor for the Ciba Foundation. Assisted by JOAN ETHERINGTON. Little, Brown and Company, Boston. 1954. xvi-333 pages. With 125 illustrations. Cloth. Price $6.75.

This Pace Is NOT Killing Us. By J. I. RODALE. Rodale Books, Inc., Emmaus, Pennsylvania. 1954. 64 pages. Price $1.00.

The Adolescent Exceptional Child. A Realistic Approach to Treatment and Training. Proceedings of the 1954 Spring Conference of the Child Research Clinic of The Woods Schools, held in New Orleans, April 9 and 10. Copies of the pamphlet are available without charge by writing to the Child Research Clinic of the Woods Schools, Langhorne, Pennsylvania.

When Minds Go Wrong. By JOHN MAURICE GRIMES, M.D., Formerly Staff Member of the Council on Medical Education and Hospitals, American Medical Association. The Devin-Adair Company, New York. 1954. ix-246 pages. Cloth. Price $3.50.

The City of Hope. By SAMUEL H. GOLTER. G. P. Putnam's Sons, New York. 1954. xii-177 pages. Illustrated. Cloth. Price $3.50.

The Reproductive System. The Ciba Collection of Medical Illustrations. Volume 2. A Compilation of Paintings on the Normal and Pathologic Anatomy. Prepared by FRANK H. NETTER, M.D. Edited by Ernst Oppenheimer, M.D. With a Foreword by John Rock, M.D., Clinical Professor of Gynecology, Harvard Medical School. Commissioned and published by Ciba Pharmaceutical Products, Summit, New Jersey. 1954. xvi-286 pages. Price $13.00.

Woman's Auxiliary . . . .

President Mrs. Maynard Emlaw, Richmond
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It's Your A.M.A.

The enthusiastic Auxiliary member says: "It's my A.M.A., too!" To be able to support her conviction with facts and to oppose successfully criticism of its activities and organizational structure, she herself must be well informed of them.

The American Medical Association was founded in Philadelphia, Pennsylvania, on May 5, 1847, as a non-profit public service institution to protect the public health and to provide the highest quality of medical care for the American people. Its purposes were to promote the science and art of medicine, to organize the medical profession and safeguard its worthy interest, and to elevate the standards of medical education and practice. Its objectives have not changed through the years.

The purpose of the Woman's Auxiliary to the American Medical Association is to assist in the program for the advancement of medicine and public health; therefore, it is essential that auxiliary members have a clear understanding of how the Association functions, its service to the profession, and its service to the general public.

Physical structure: The modern nine-story office building at 535 North Dearborn Street, Chicago, is the control center of A.M.A.'s activities. Here 900 employees carry on the work of the Association's 54 councils, committees, bureaus and departments.

A.M.A.'s representational structure is similar to that of the United States. Policies are determined by a House of Delegates composed of elected representatives of the state and territorial medical associations, the scientific sections and appointed representatives of federal medical services.

Services to the profession

The American Medical Association:

(1) Provides postgraduate education through its annual and clinical educational meetings and its weekly medical journal and nine monthly specialty journals.

(2) Assures good medical education and hospital training by requirement of certain high standards.

(3) Maintains a national doctor placement service.

(4) Provides assistance on medico-legal problems, and through the Washington office represents the profession in our national government.

(5) Publishes a Directory which lists, with biographical material, the legally qualified physicians in the United States and Canada; publishes the Standard Nomenclature which is the classification of accepted terminology used by 70% of the nation's hospitals for disease and operation index filing; publishes the New and Non-official Remedies which lists articles and preparations found acceptable under rules of Council on Pharmacy and Chemistry.

(6) Encourages ethical medical practice through emphasis on local, state and national levels of an accepted code of ethics, the A.M.A. Principles of Ethics.

Annual dues are $25.00; of this, $15.00 is for the Journal subscription and the remaining $10.00 is used for the countless many other activities.

Service to the Public

Some of the ways in which A.M.A. aids the public are:

(1) By evaluating drugs, special dietetic foods, and therapeutic and diagnostic devices as to reliability, proven therapeutic value and limitations.

(2) The Committee on Cosmetics evaluates cosmetics on basis of set standards.

(3) The Committee on Pesticides promotes coordination of research and medical activities involving use of insecticides, rodenticides, herbicides.

(4) Council on Foods and Nutrition encourages good practices in food advertising and labeling and provides factual information about the importance of nutrition in combating disease.

(5) A special bureau investigates and disseminates information on quacks, medical fads, secret remedies and various phases of pseudo-medicine.

Presented at the Eleventh Annual Conference of State Presidents, Presidents-Elect and National Committee Chairmen, Woman's Auxiliary to the American Medical Association, held November 16-18, 1954 at the Drake Hotel, Chicago.

(6) Publishes "Today's Health" which provides authentic health information to the lay reader.

These are but a few of the services provided to the physician and to the general public. In addition, the Chicago office gives willing assistance to auxiliary members whenever called upon.

The word "auxiliary" is defined as meaning *to influence, to assist,* and *to support,* and the members of the Woman's Auxiliary to the American Medical Association will be better able to fulfill these functions when they are accurately informed as to what our A.M.A. stands for.

LUCIA K. EMLAW (Mrs. Maynard R.)
President, Woman's Auxiliary to the Medical Society of Virginia

Correspondence . . . .

Incidence of Goiter

To The Editor:

The recent paper by Philips on the "Incidence of Goiter in Southwest Virginia (Virginia Medical Monthly, 81, 386-7, 1954) prompts me to recall to your readers that goiter was once common in certain places in that region.

In 1831, Dr. John T. Smith wrote (Gibson, W., Institutes and Practice of Surgery, 8th edition, Philadelphia, 1850, Vol. 2, p. 61):

"At King's Salt Works, in the county of Washington, Virginia, and not far from my own residence this disease has prevailed for a number of years; . . . Persons living at the distance of a half, or even quarter of a mile are not subject to the disease.

"Contrary to what might be supposed from the aspect of the neighboring county, this disagreeable affliction is of exceedingly rare occurrence in this part of the state, with the exception above alluded to . . . every woman who goes to King's Saltworks to live, previously makes up her mind to become sooner or later a subject of goiter."

Some years later, the disease seems to have become more general and as frequent as in Ohio at that time. In 1850, Daniel Drake wrote from Wytheville (Western Lancet, 11, 558-569, 1850):

"*Goiter.* Cases are scattered here and there. I think they are not more numerous, in proportion to the population, than in the valleys of the Scioto and Cuyahoga."

It would be interesting to know if goiter is still common in Saltville or in the vicinity of Wytheville and, if not, if there is any information as to when the change took place.

I should appreciate hearing from any of your readers who have any information on this subject.

ISADOR GREENWALD
Professor Emeritus, New York University College of Medicine
550 First Avenue
New York 16, N. Y.
December 10, 1954

President's Message . . . .

The Cost of Medical Care

ALL of us know without reference to statistics that the cost of medical care has increased to such an extent that an ordinary illness can be catastrophic to an ordinary family and the cost of care of medical indigents has become a terrific burden to the taxpayer. The medical profession can do more to alleviate this steadily increasing expenditure of money than any other group, and I am afraid we are not half trying.

It is obvious that many modern and expensive therapeutic agents have saved many lives, have rehabilitated many invalids, and in spite of the initial high cost have actually reduced the total expenditure. It is also obvious that the routine and, much too often unnecessary, use of these drugs has added tremendously to the total cost. Furthermore, when the older preparations have been produced in quantity and the price has become reasonable, the doctor much too often abandons them for a new expensive one when the old one would do just as well.

It is evident too that there is profligate use of laboratory procedures. Perhaps the greatest waste of money is in unnecessary x-ray examinations. It seems almost routine for the doctor to minimize his ability to diagnose pulmonary conditions when he can learn so much from x-ray pictures. It should not be forgotten, however, that there are many lung and other changes in the body which are not revealed by roentgen studies. The same applies to many other physical and biochemical procedures.

Finally it is obvious that there is much unnecessary hospitalization of patients. Some of the increase in hospital patients is justified by the changes that have developed in the average modern home, but a large proportion is due to indulgence of the doctor, particularly the various specialists, to his own and the patient's convenience. Many in this latter group have hospital insurance and this has resulted in greater increases in the rates for such coverage. A large portion of the hospital care of indigent patients is in the hospitals of our teaching institutions, these patients have a much longer average stay than private patients, some of this is justified, but a considerable reduction in this field should be made. The waste and unnecessary use of expensive material in all hospitals offer another field for major economy.

The cost of living has become terrific, this cost of keeping our people alive and healthy can be materially reduced by the members of The Medical Society of Virginia.

Carrington Williams

President

Editorial . . . .

Cortisone and ACTH

SIX years have passed since the dramatic appearance of cortisone and ACTH on the medical scene. The initial striking success of these agents in treating rheumatoid arthritis was soon followed by similarly spectacular results in many other diseases and enthusiasm ran high. A note of caution was introduced as various undesirable and sometimes serious side effects were encountered. Then it became increasingly apparent that continued treatment was necessary to maintain improvement in most conditions treated. Enthusiasm waned and a note of pessimism appeared. The statement that cortisone and ACTH "do not cure anything" sounded shocking at first but then its full truth sank in. What can we say of these agents today? Are they the priceless boon to the sufferer that we once thought, or are they treacherous drugs to be avoided except as a last resort? Elsewhere in this issue there is presented a symposium on the treatment of asthma. Undoubtedly the use of cortisone and ACTH is referred to in these papers. It seems appropriate at this time to review some of the facts about these agents.

ACTH or adrenocorticotropic hormone is secreted by the anterior lobe of the pituitary gland and stimulates the adrenal cortices to greater output of adrenocortical hormone (ACH). The exact nature of ACH is not known but it appears to consist of a combination of steroids which may all be derived from a single parent compound. Cortisone (compound E) and hydrocortisone (compound F) are two steroids which have been isolated from the adrenal gland, which have potent effects practically identical with those of adrenocortical hormone. Commercial ACTH is prepared from the pituitary glands of animals, but cortisone and hydrocortisone are both made synthetically.

Among the many effects of adrenocortical hormone the most important appears to be its ability to block the effect of various injurious agents on body cells and thus to protect the cells from damage. This constitutes an integral part of the total body defense mechanism. In time of stress or danger the brain is informed of the need for extra protection and stimulates the pituitary to discharge more ACTH, which whips up the adrenal glands to greater output of ACH. Thus the body cells are fortified to better withstand the danger. Whenever the blood level of ACH rises too high, this exerts a suppressive effect on the pituitary production of ACTH, and the balance is restored. Thus an optimum amount of circulating adrenocortical hormone is assured in health and in disease. There are states of continuously excessive production of ACH, or hypercorticoidism, which give rise to the clinical picture known as Cushing's syndrome. Inadequate ACH output, or hypocorticoidism, is exemplified in Addison's disease.

The use of cortisone or hydrocortisone in Addison's disease is a form of substitution therapy comparable to giving insulin to a diabetic. A hypocorticoid individual is rendered eucorticoid. Dosage is small, usually 12.5 mg. to 25 mg. daily. There are no side effects and the patient's well being is markedly restored. Similarly ACTH may be given in small amounts in pituitary insufficiency with secondary adrenal failure, to restore normal adrenal activity. In actual usage the necessity for continued injections makes ACTH impractical, and cortisone substitution treatment is usually preferred even in pituitary failure.

The major use of cortisone and ACTH, however, is in treating a large group of diseases which include rheumatoid arthritis, other collagen diseases, asthma, allergies, disorders of the skin and eye, and a variety of other maladies. In none of these conditions is any adrenal deficiency present. Cortisone and ACTH are not given as substitution therapy, but in large doses with the deliberate aim of producing a state of

hypercorticoidism. The beneficial effect on the disease being treated depends entirely on the degree of hypercorticoidism produced. The noxious agent which is causing the disease is unaffected, and indeed may be entirely unknown, but its harmful effect on the cells of the body is blocked and the signs and symptoms of the disease are abolished. Thus pneumonia may be abolished while pneumococci flourish in the lungs, and asthma disappears while hypersensitivity may remain. The clinical improvement is purchased, however, at the cost of continuing hypercorticoidism, and any or all of the signs of Cushing's syndrome may be expected if treatment is prolonged.

The disadvantages of this type of treatment are readily apparent. First the cause of the disease being treated is not removed, and unless it is by nature self limited, the disease will surely recur when cortisone or ACTH is stopped. This necessitates indefinitely continued treatment in most cases. Secondly the maintenance of a continued state of hypercorticoidism itself constitutes a considerable hazard. The danger of producing diabetes or hypertension is not as great as was once feared, but more subtle dangers lie in breaking down resistance to the spread of tuberculosis or other infection, in favoring the development of peptic ulcer, in increasing the work of the heart and kidneys by salt loading, and, finally, in the masking of various complications which may develop. The third disadvantage is the suppression of the patient's own adrenal glands when cortisone is given, and the suppression of pituitary responsiveness to stress when ACTH is given. When prolonged treatment with either ACTH or cortisone has been given, cessation of treatment is inevitably followed by a period of hypocorticoidism which may last for weeks or months. Exposure to stress during this period may be fatal unless protective cortisone is reinstituted. Neither tapering off of cortisone dosage nor the administration of ACTH before stopping cortisone will fully obviate this danger.

What can we say about the use of a type of therapy as unphysiologic and as potentially dangerous as this, where one disease is produced to relieve another? We can say first that cortisone or ACTH treatment should never be undertaken lightly. These agents should be given only in diseases that are likely to be otherwise fatal or so disabling as to incapacitate the patient, and only when all other types of treatment have been exhausted. Thus we would consider it unwise to use cortisone for relief of occasional bouts of a thing, but would give cortisone or ACTH to a patient in intractably status asthmaticus where all other remedies had failed. We would not use cortisone in early rheumatoid arthritis, but would consider it justified in advanced and crippling cases. The dose should be kept as low as possible, with the aim of making pain bearable rather than of total relief of all symptoms. In treating certain usually fatal conditions such as pemphigus, or disseminated lupus erythematosus, such large doses may be necessary to control the disease that a definite Cushing's syndrome may be inevitable. This is justified if we are thereby keeping the patient alive. Testosterone, salt restriction and added potassium may help alleviate some of the features of the Cushing's syndrome.

Before beginning cortisone or ACTH in any condition, a physician must look ahead to the future and must assume responsibility for either the continuation of treatment or the necessary precautions and long period of after-care when the drug is withdrawn. In treating a condition that is by nature self limited, such as acute rheumatic fever, or acute exfoliative dermatitis, it is hoped that a limited course of treatment may be given and the drug then withdrawn. Or in treating a disease that may show spontaneous remissions, such as the early stage of disseminated lupus erythematosus, there is still hope that the drug may later be omitted and given only for exacerbations. But in treating a disease that is by nature chronic, a permanent program of cortisone must be planned and the problem is to keep the dosage to the minimal amount compatible with reasonable control of the disease. The physician should familiarize himself with

the various routes of administration and dosage forms of cortisone, hydrocortisone, and ACTH. He should be familiar with the various phenomena of the hypercorticoid state and alert to the warning signs of serious trouble. He must know the contraindications to the use of these agents, such as cardiac or renal failure, peptic ulcer and tuberculosis. He must understand that pre-existing diabetes or hypertension may be aggravated, and that unfavorable reaction are likely in patients of neurotic or unstable temperament.

It should be stated that the above remarks pertain chiefly to the systemic use of these agents, oral or parenteral. Local or topical use of cortisone or hydrocortisone may be effective without producing any general state of hypercorticoidism. It should be constantly remembered, however, that even local effects depend on the blocking action of the drug, and in no way affect the cause of the condition being treated.

Indiscriminately used, these potent drugs can do untold harm. But with proper knowledge and judgment the physician may use them confidently and with gratifying results. They still represent one of the major therapeutic accomplishments of our age.

H. St. George Tucker, Jr., M.D.

Society Proceedings . . . .

Rockingham County Medical Society.

New officers of the Rockingham County Medical Society for 1955 are: President, Dr. B. W. Nash, Timberville; vice-president, Dr. Jacob Huffman, Dayton; and secretary-treasurer, Dr. William J. Hotchkiss, Broadway.

Medical Society of Northern Virginia.

At the meeting of this Society on December 14th, the following officers were elected: President, Dr. F. L. Hopewell, Strasburg; vice-president, Dr. H. P. Macculbin, Winchester, and secretary-treasurer, Dr. Dennis P. McCarty, Front Royal.

Williamsburg-James City Medical Society.

The following new officers were elected at the meeting on December 8th: President, Dr. E. B. Kilby, Toano; vice-president, Dr. Carlton J. Casey, Williamsburg, and secretary-treasurer, Dr. Linwood Earley, Williamsburg. They were installed on January 12th.

Virginia Peninsula Academy of Medicine.

Officers for the Academy for 1954-1955 are: President, Dr. O. W. Ward, Sr., Hampton; vice-president, Dr. Robert Peirce, Newport News, and secretary-treasurer, Dr. William T. Watkins, Jr., Newport News.

The Fredericksburg Medical Society

Has elected Dr. Joseph MacKnight as president for 1955. Dr. John B. Rose is vice-president, and Dr. H. Lee Earthardt, secretary-treasurer.

Arlington County Medical Society.

The following will serve as officers of this Society for 1955: President, Dr. H. C. Bate, Jr.; vice-president, Dr. Hermann J. Diamant; secretary, Dr. Stephen J. Sheehy; treasurer, Dr. T. A. McGavin; and Member-at-large, Dr. J. R. B. Hutchinson.

Fourth District Medical Society.

Officers elected at the meeting on December 14th are: President, Dr. William B. McIlwaine, Petersburg; vice-presidents, Drs. Charles W. Scott, Burkeville, and Robert D. Keeling, South Hill; secretary-treasurer, Dr. John G. Graziani, Farmville, and Chairman of Steering Committee, Dr. J. B. Kiser, Emporia.

The Southwestern Virginia Medical Society

Will hold its next meeting in Radford on Thursday, April 14th. Dr. C. C. Hatfield, Saltville, is president, and Dr. Reverdy H. Jones, Jr., Roanoke, secretary-treasurer.

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News . . . .

Calendar of Coming Events

ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE—Palmer House, Chicago, February 5-8

INTERNATIONAL COLLEGE OF SURGEONS—Mid-Atlantic Division Regional Meeting—Statler Hotel, Washington, D. C., February 11-12

TRI-STATE MEDICAL ASSOCIATION—Hotel Chamberlin, Old Point, Va., February 21-22

NATIONAL CONFERENCE ON RURAL HEALTH—Schroeder Hotel, Milwaukee, Wis., February 24-26

AUTO MEDICAL ASSOCIATION—Hotel Statler, Washington, D. C., March 20-23

INTERNATIONAL ACADEMY OF PROCTOLOGY—The Plaza Hotel, New York City, March 24-26

EASTERN STATES EDUCATION CONFERENCES—New York Academy of Medicine, New York City, April 21-22

VIRGINIA SOCIETY OF OPHTHALMOLOGY & OTOLARYNGOLOGY—Natural Bridge, Va., May 6-7

VIRGINIA ACADEMY OF GENERAL PRACTICE—Hotel Chamberlin, Old Point, Va., May 13-15

Richmond Committee on Arrangements.

The following are members of the Committee on Arrangements for the Annual Meeting of The Medical Society of Virginia to be held in Richmond, October 16-19:

General Chairman—Dr. John P. Lynch
Scientific Exhibits—Dr. Frank M. Blanton
Technical Exhibits—Dr. Oscar Hite
Press and Publicity—Dr. William H. Higgins, Jr.
Hotels and Halls—Dr. W. Linwood Ball
Entertainment—Dr. William F. Grigg, Jr.
Golf—Dr. C. C. Chewning, Jr.
Auxiliary—Mrs. J. R. Grinels and
Mrs. George H. Snead

Officers of Speciality Societies.

Specialty groups which met in conjunction with the First Interstate Scientific Assembly in Washington, October 31-November 5, elected the following officers:

Virginia Pediatric Society
President—Dr. E. L. Kendig, Jr., Richmond
Secretary—Dr. William Spencer, Richmond
Virginia Society of Anesthesiologists
President—Dr. W. E. Pembleton, Richmond
Secretary—Dr. John Rosenthal, Norfolk
Virginia Orthopedic Society
President—Dr. Allen M. Ferry, Arlington
Secretary—Dr. William Deyerle, Richmond
Virginia Society for Pathology
President—Dr. Edward Levy, Norfolk
President-elect—Dr. John Hooker, Danville
Secretary-Treasurer—Dr. George J. Carroll, Suffolk
Virginia Neuropsychiatric Society
President—Dr. Granville L. Jones, Williamsburg
Vice-President—Dr. Frank Strickler, Roanoke
Secretary-Treasurer—Dr. George Fultz, Richmond
Virginia Obstetrical and Gynecological Society
President—Dr. Eugene S. Groseclose, Roanoke
President-elect—Dr. George S. Hurt, Roanoke
Secretary-Treasurer—Dr. Chester D. Bradley, Newport News
Virginia Section, American College of Physicians
Chairman—Dr. Alex F. Robertson, Staunton
Secretary-Treasurer—Dr. James F. Waddill, Norfolk
Virginia Radiological Society
President—Dr. George Cooper, Charlottesville
President-elect—Dr. Allen Barker, Roanoke
Secretary-Treasurer—Dr. P. B. Parsons, Norfolk
Virginia Diabetes Association
President—Dr. Thomas S. Edwards, Charlottesville
Vice-Presidents—Dr. William A. Read, Newport News
Dr. Collier W. Salley, Norfolk
Secretary-Treasurer—Dr. James F. Conner, Kecoughtan

Dr. Carrington Williams, Sr.,

President of The Medical Society of Virginia, has received an additional honor by his election as President of the Southern Surgical Association at its annual convention held at Hollywood Beach, Florida in December. He has been treasurer of the Association for ten years.

Dr. W. Taliaferro Thompson, Jr.,

Richmond, has been named chief of medical services at McGuire Veterans Administration Hospital and promoted to associate professor of medicine at the Medical College of Virginia. At McGuire's he succeeds the late Dr. John Powell Williams.

Lynchburg General Hospital Staff.

Dr. Holcombe Hurt has been elected chief of staff of the Lynchburg General Hospital, succeeding Dr. L. R. O'Brian, Jr. Dr. J. E. Warren was named assistant chief and Dr. Donald Shotton was elected as secretary.

Dr. Kyle Mundy was elected as chief of surgical services, succeeding Dr. Macey Rosenthal.

Dr. Dennis P. McCarty

Has been elected president of the Front Royal-Warren County Chapter, Virginia Division, of the American Cancer Society.

The American Academy of General Practice.

Thirty two of the nation's top medical authorities will participate in the program of the Seventh Annual Scientific Assembly in Los Angeles, March 28-31. Among the speakers are: Dr. Alton Ochsner, New Orleans; Dr. Digby Leigh, Los Angeles; Dr. Hugh H. Hussey, Washington D. C.; Dr. William Sodeman, University of Missouri; Dr. Lowell Goin, Los Angeles; Dr. Charles Larson, Tacoma; and Dr. Edward Weiss, Philadelphia.

There will be panel discussions on diabetes, childhood disasters, peripheral vascular diseases, neurology, endocrinology, handling athletic injuries and preserving the doctor's life. Other subject material that will be covered includes newer concepts of allergy therapy, anesthetic emergencies, treatment of the ambulatory fracture case, medical aspects of crime detection, using the laboratory in liver disease and the negative roentgenogram.

Scientific exhibits will supplement the formal lecture program.

Bermuda-Nassau Cruise.

An eight-day cruise to Bermuda and Nassau has been arranged for physicians and their wives following the A.M.A. meeting at Atlantic City in June. The party will sail from New York on June 10th aboard the Furness Line steamer Ocean Monarch. The ship docks at St. George's, Bermuda early Sunday morning, sailing again on Monday at 4 P.M. They will reach Nassau on Wednesday, leaving at midnight and docking in New York on Saturday, June 18th.

All space is being held for the A.M.A. and reservations should be made immediately. Contact W. M. Mehoney, Chicago, Burlington and Quincy Railroad, 105 West Adams Street, Chicago.

Professor of Surgical Cardiology.

Dr. John Francis Dammann, Jr., has been appointed as associate professor of surgical cardiology in the department of surgery of the University of Virginia School of Medicine. He has worked intimately with Dr. William H. Muller, Jr., newly appointed chairman of the department of surgery over a period of six years, both at Johns Hopkins and the University of California, where Dr. Dammann has been assistant professor of pediatrics since 1950.

Conference on Crippled Children.

The Fourth Annual Conference on Crippled Children will be held March 2-3 at the Hotel Roanoke, Roanoke. This conference is sponsored each year by the Virginia Council on Health and Medical Care at the request of The Nemours Foundation of Wilmington, Delaware. The theme for this conference is "The Emotionally Disturbed Child" with Dr. Adelaide Johnson, child psychiatrist at the Mayo Clinic, and Dr. Samuel A. Kirk, Director, Institute for Research on Exceptional Children, University of Illinois, as the two main speakers. There will be a panel on Care and Treatment of the Emotionally Disturbed Child and one on Meeting the Needs of the Emotionally Disturbed Child in Virginia.

All physicians are cordially invited to attend the conference. For further details, address the Virginia Council on Health and Medical Care, 102 E. Franklin Street, Richmond.

For Sale

Modern office equipment, complete, including fluoroscope and electro-cardiograph and practice in Southside Virginia city. Excellent hospital facilities. Write Box #40, care the Monthly, Box 5085, Richmond 20, Va. (Adv.)

Wanted.

A physician for contract practice in coal camp. Write #30, care Virginia Medical Monthly, Box 5085, Richmond 20, Va. (Adv.)

Office and Equipment Available.

The office is located in Newport News, near the shipyard, and is fully equipped for private practice, with reception room, laboratory, consultation and examining rooms, and private bath. All the surgical instruments, operating equipment and laboratory apparatus will be appraised to be sold at a reasonable price. Doctor recently deceased. For full information contact Mrs. James E. Marable, 525 64th Street, Newport News. (*Adv.*)

Physician Wanted

For community of 1100 in central Virginia. For full information, write #35, care the Virginia Medical Monthly, P. O. Box 5085, Richmond 20, Virginia (*Adv.*)

Residency Available.

A residency in otolaryngology at the University of Virginia, Charlottesville, is available July 1, 1955. Training is also given in maxillo-facial surgery and broncho-esophagology. For additional information, contact the Otolaryngologist in Chief, University of Virginia Hospital, Charlottesville, Virginia. (*Adv.*)

For Sale.

Best offer will take an electric Scanlan-Morris autoclave and an unused St. Luke's delivery bed. Must sell at once because of storage problem. A Lumetron electrocolorimeter, model 401A, in excellent condition for $150.00 and will guarantee for one year. Other miscellaneous equipment. Contact R. H. Ragland, 208 Richelieu Ave., S. W., Roanoke, or phone 25325. (*Adv.*)

Obituaries . . . .

Dr. Henry Adams Tabb,

Prominent physician of Gloucester, died January 7th following a heart attack. He was a native of Gloucester County and sixty-seven years of age. Dr. Tabb taught school for several years before he entered the Medical College of Virginia, from which he graduated in 1913. He had practiced in Gloucester since that time. Dr. Tabb had been an active member of The Medical Society of Virginia for forty years. At the time of his death, he was chairman of the Walter Reed Commission, the chief duty of which is the maintenance of the Walter Reed Home, "Belroi", located in Gloucester County. His wife survives him.

Dr. James Edward Marable,

Well known physician of Newport News, died November 27th, following a heart attack. He was a native of Isle of Wight County and sixty years of age. He received his medical degree from the University of Virginia in 1918 and had practiced in Newport News since that time. Dr. Marable was in the service during World War II, serving as Lieutenant Commander at the Great Lakes Training Station and the Brooklyn Navy Yard. As a Captain he served at an Oregon hospital, before returning to his practice in Newport News. For the past two years, Dr. Marable has been on the medical staff of the Newport News Shipbuilding and Dry Dock Company. He also served as a staff member at Mary Immaculate and Riverside Hospitals. Dr. Marable had been a member of The Medical Society of Virginia for thirty-four years. His wife, two daughters and a son survive him.

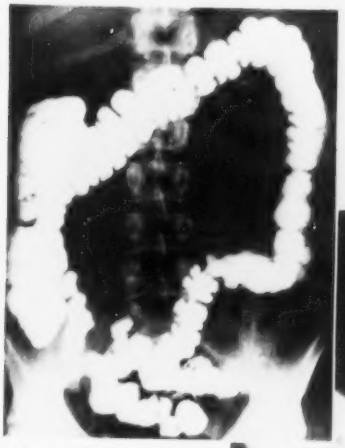
Dr. Frederick Oliver Fay,

Prominent Norfolk physician, died November 15th, following a heart attack. He was fifty years of age and a graduate of the Medical College of Virginia in 1930. Dr. Fay was an eye, ear, nose and throat specialist and had practiced in Norfolk since 1940. He was in service during World War II, serving as Lieutenant Commander and later as Captain at the Naval Operating aBse at Norfolk and at San Juan, Puerto Rico. Dr. Fay was official ophthalmological consultant to the Norfolk City Venereal Disease Clinic. He was a member of many civic and fraternal organizations. Dr. Fay had been a member of The Medical Society of Virginia since 1941. Two sons survive him.

Dr. James Thomas Shelburne,

Critz, died December 25th, at the age of sixty-five. He was a graduate of the Medical College of Virginia in 1915. Dr. Shelburne was in the Service during World War I, following which he practiced for a while in West Virginia. He located at Critz in 1921 and also had an office at Martinsville. Dr. Shelburne had been a member of The Medical Society of Virginia for thirty years. A daughter survives him.

METAMUCIL® IN CONSTIPATION



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Ulcerative Colitis

Atonic Colon

Smoothage in Correction of Colon Stasis

To initiate the normal defecation reflex, the "smoothage" and bulk of Metamucil provide the needed gentle rectal distention.

Once the habit of constipation has been established, due to any of a large number of causes, it becomes a major problem. Self-medication with irritant or chemical laxatives, or repeated enemas, usually causes a decreased, sluggish defecation reflex and may result in its complete loss.

Rectal distention is a vital factor in initiating the normal defecation reflex, and sufficient bulk is thus of obvious importance in restoring this reflex. Metamucil provides this bulk in the form of a smooth, nonirritating, soft, hydrophilic colloid which gently distends the rectum and initiates the desire to evacuate. Metamucil demands extra fluid, imparting even greater smoothage to the intestinal contents.

It is indicated in chronic constipation of various types—including distal colon stasis of the "irritable colon" syndrome, the atonic colon following abdominal operations, repressions of defecation after anorectal surgery and in special conditions such as the management of a permanent ileostomy. Metamucil is the highly refined mucilloid of Plantago ovata (50%), a seed of the psyllium group, combined with dextrose (50%) as a dispersing agent.

The average adult dose is one rounded teaspoonful of Metamucil powder in a glass of cool water, milk or fruit juice, followed by an additional glass of fluid if indicated.

Metamucil is supplied in containers of 4, 8 and 16 ounces. It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

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RADFORD, VIRGINIA

125-BED PRIVATE PSYCHIATRIC HOSPITAL FOR THE TREATMENT OF NERVOUS AND MENTAL DISORDERS, INCLUDING ALCOHOLISM AND ADDICTION.

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James P. King, M. D., Director

James K. Morrow, M. D. Thomas E. Painter, M. D. Daniel D. Chiles, M. D.

James L. Chitwood, M. D., Medical Consultant

Affiliated Clinic Offering Psychiatric and Psychological Evaluation and Therapy:

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1400 Bland Street Bluefield, W. Va.

David M. Warne, M. D., Director

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Richmond, Virginia

A private hospital accepting for diagnosis and treatment organic neurological conditions, selected psychiatric and alcoholic cases, metabolic disturbances of an endocrine nature, individuals who are having difficulty with their personality adjustments, and children with behavior problems. Patients with general medical disorders admitted for treatment under our staff of visiting physicians

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A Modern Hospital for the Treatment of Alcoholism Exclusively

- A private hospital offering scientific, institutional, medical, psychological, reflex, reduction and other methods for the rehabilitation of consent patients suffering from alcoholism.

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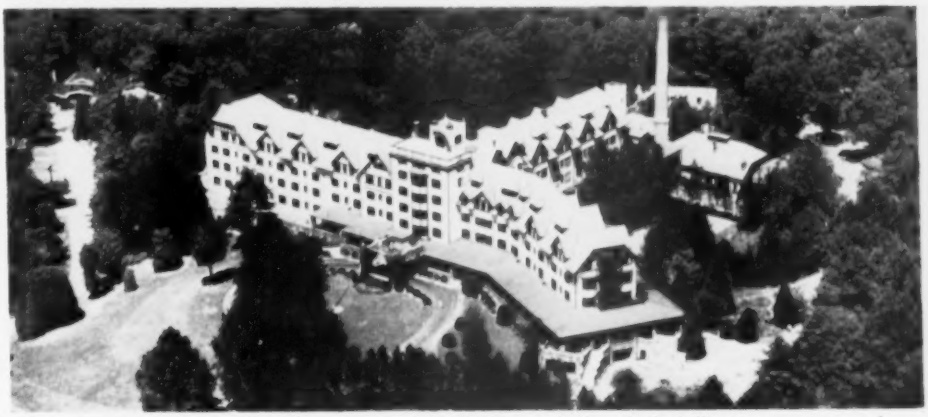

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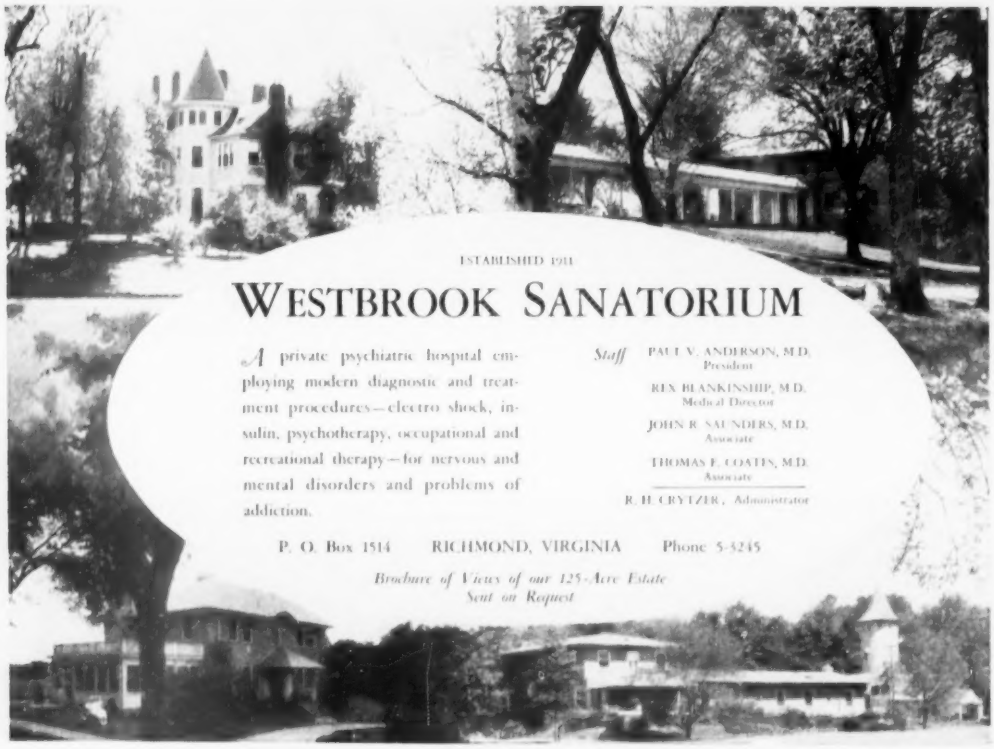

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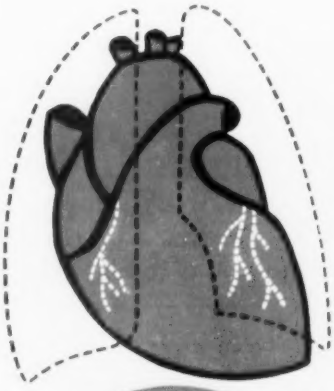

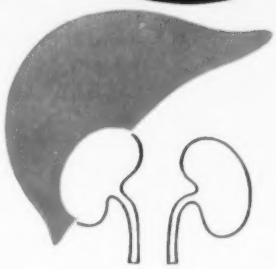

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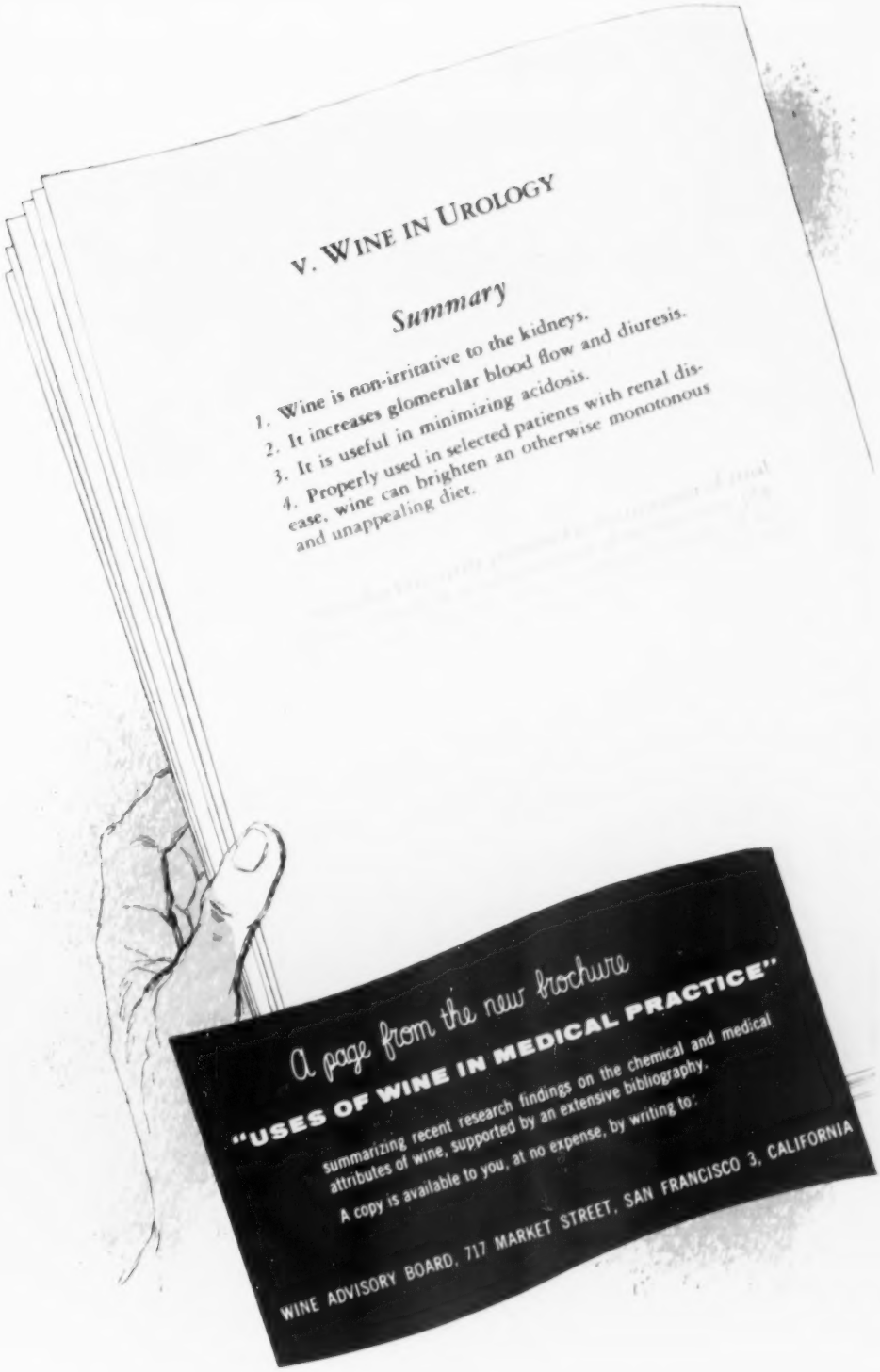
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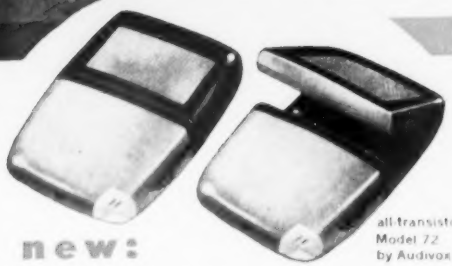

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PROGRAM

Monday, February 21

Surgery of Arteries	William H. Muller, M. D.
Surgery of Heart	Thomas N. P. Johns, M. D.
Surgery of Hypertension	Keith Grimson, M. D.
Intermission	
Treatment of Peripheral Vascular Disease	E. L. Lowenberg, M. D.
Cancer of the Brain: Newer Methods of Diagnosis and Treatment	J. E. Watts, M. D.
Lunch	
Glaucoma in Geriatrics	Rudolph Thomason, M. D.
Congenital Heart Disease	Dale Groom, M. D.
Drug Therapy of Hypertension	Herbert G. Langford, M. D.
Intermission	
Coronary Disease	James L. Camp, III, M. D.
Cardiac Emergencies in General Practice	J. L. Hamner, M. D.

Tuesday, February 22

Disease of the Lung	Dean B. Cole, M. D.
Diseases of the Humerus	Fay A. Carmines, M. D.
The Evaluation of the Sterile Couple	Robert A. Ross, M. D.
Geriatric Gynecology	Waverly R. Payne, M. D.
Intermission	
Clinical Pathological Conference	Benjamin Baker, M. D.
Lunch	
An Intermediate Hospital Unit for the Care of the Patient with Long Term Illness	James Conner, M. D.
Prognosis with Early vs Delayed Diagnosis and Treatment of Diabetes	Charles W. Styron, Sr., M. D.
Surgical Lesions of the Spinal Cord Simulating Non Surgical Conditions with a Report of Ten Cases	John Meredith, M. D. Charles Troland, M. D.
The Early Treatment of Severe Full Thickness Burns	Herrick Thomas, M. D.
Precocious Pregnancy	Charles W. McCants, M. D.

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The State Board of Medical Examiners of Virginia

The next meeting of the Board will be held at the Richmond Hotel, Richmond, Virginia, June 15, 1955, and the examinations will be held at the same hotel June 16 to 18, inclusive. June 1st is the deadline for receipt of applications for the examination and documents and papers to be discussed and acted upon by the Board. The secretary of the Board is Dr. K. D. Graves, 631 First Street, S. W., Roanoke, Virginia.

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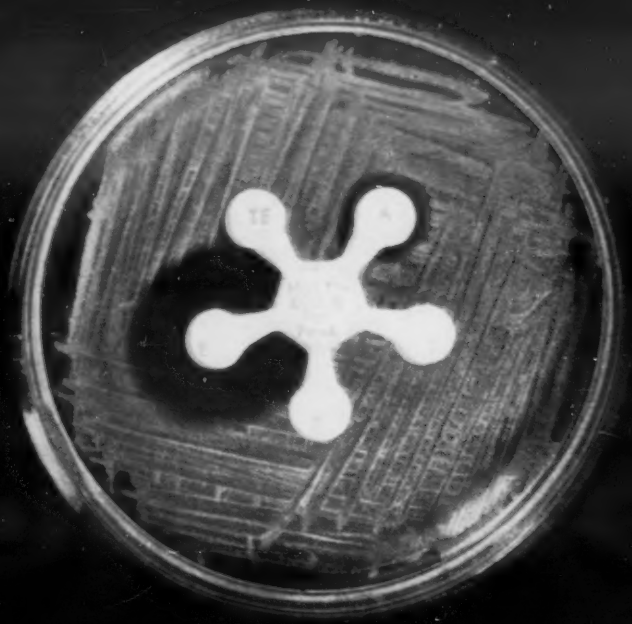

This is an actual strain of *staphylococcus aureus*, isolated from a 5-week-old infant. Note extreme sensitivity of the organism to ERYTHROCIN—although it easily resists penicillin and three broad-spectrum antibiotics. This organism may be associated with *sinusitis* . . . *otitis media* . . . *tonsillitis* . . . *abscess* . . . *bronchopneumonia* . . . *empyema* . . . *carbuncle* . . . *pyoderma* . . . *bronchiectasis* . . . *furunculosis* . . . *pharyngitis* . . . *septicemia* . . . and *tracheobronchitis*.

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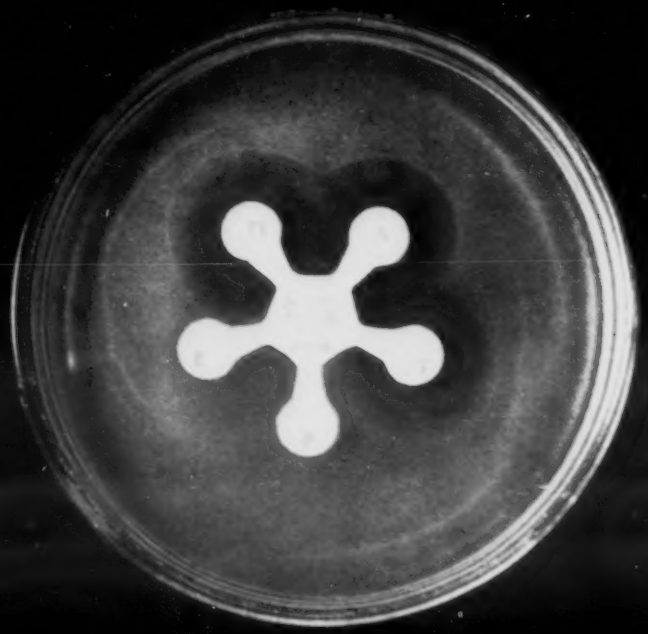
(ERYTHROMYCIN STEARATE, ABBOTT)

Against common intestinal flora



This sensitivity test shows ERYTHROCIN, penicillin and three broad-spectrum antibiotics against a typical strain of *E. coli*. Note that ERYTHROCIN and penicillin do not affect growth of the organism—*while all three broad-spectrum antibiotics show marked inhibitory action*. Since ERYTHROCIN is inactive against gram-negative organisms, it is less likely to cause alteration in common intestinal flora—with an accompanying low incidence of side effects.

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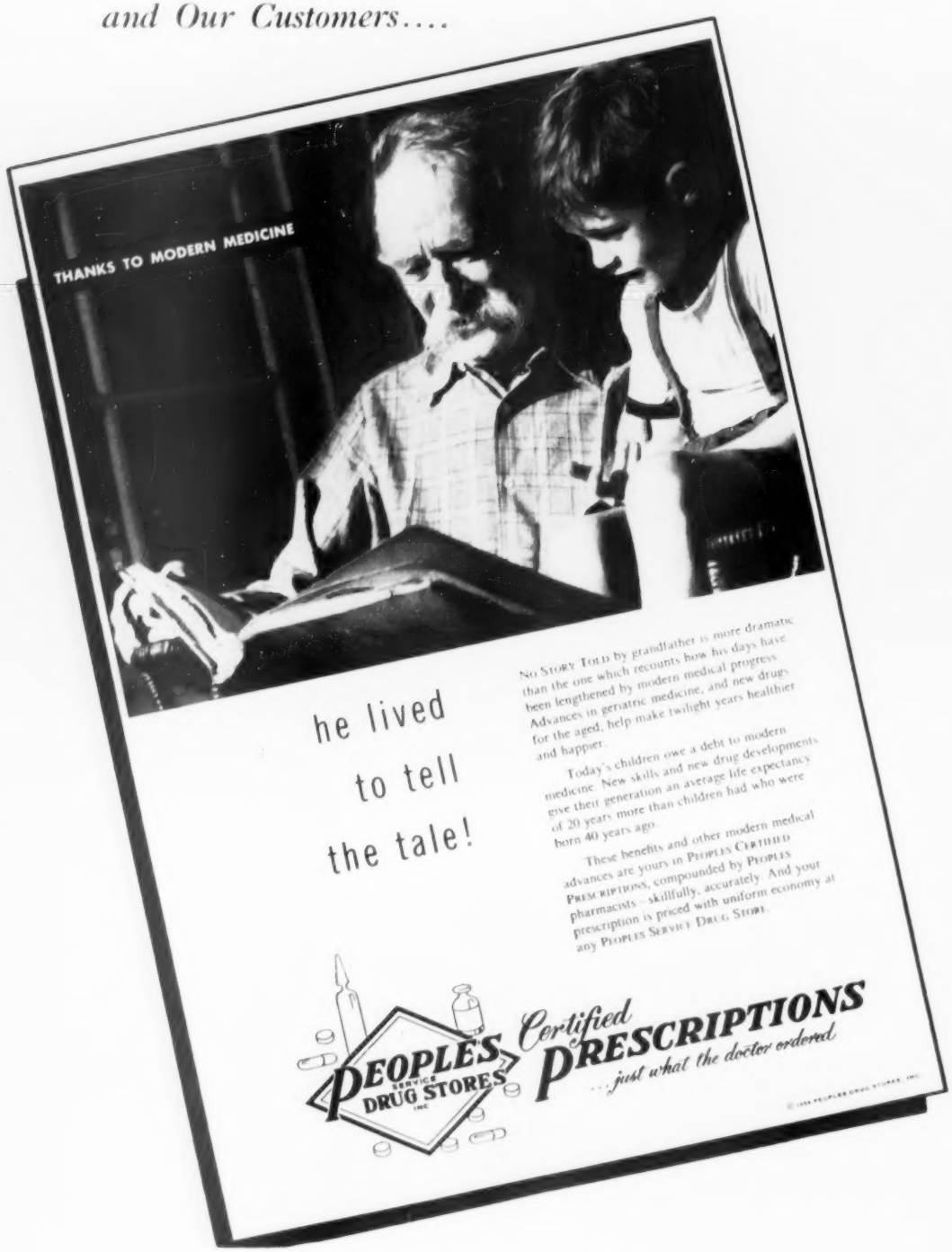
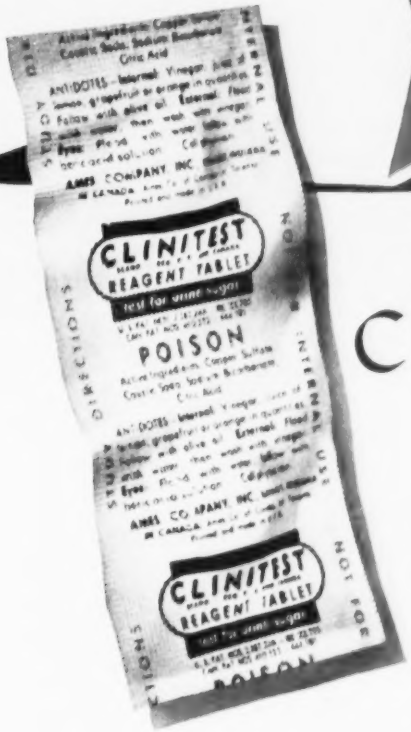


the last tablet as accurate as the first



sealed-in-foil

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REAGENT TABLETS

a rapid, reliable urine-sugar test every time because every batch of *Clinitest* Sealed-in-Foil Reagent Tablets is tested for stability under conditions as exacting as a tropical rainy season—86° to 90° temperatures and 95% humidity.

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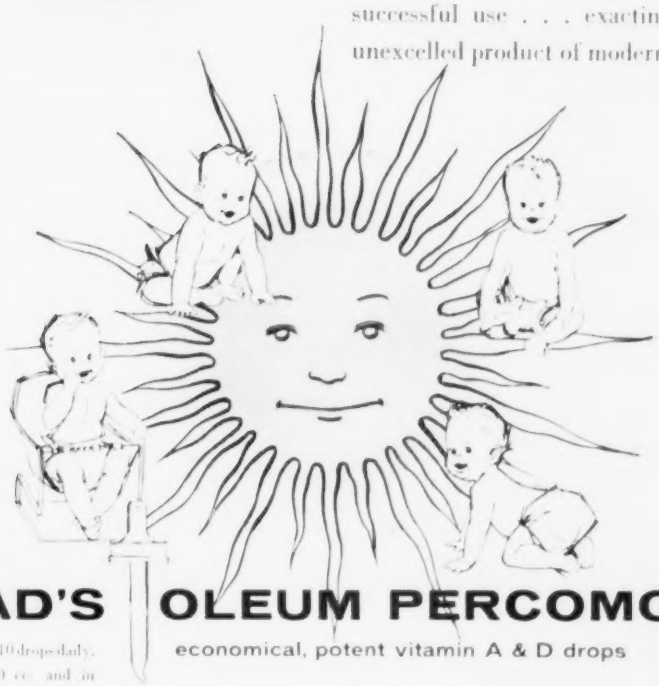
Mead's Oleum Percomorphum is a potent, effective source of vitamins A & D . . . that can be given at a cost of about a penny a day.

Specify Mead's Oleum Percomorphum for utmost dependability. It assures your pediatric patients a vitamin preparation with more than 20 years of successful use . . . exactingly assayed . . . an unexcelled product of modern processing.



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